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A

PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART

AND

GREAT VESSELS.

BY THE SAME AUTHOR.—(Just Issued.)

A PRACTICAL TREATISE

ON THE

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INCLUDING THE

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BLANCHARD & LEA, ***Philadelphia.***

A

PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART

AND

GREAT VESSELS,

INCLUDING

THE PRINCIPLES OF PHYSICAL DIAGNOSIS.

BY

WALTER HAYLE WALSHE, M.D.,
FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;
PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE IN UNIVERSITY COLLEGE, LONDON; PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL; CONSULTING PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION.

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TO

P. C. A. LOUIS, M.D.

CONSULTING PHYSICIAN TO THE HÔTEL-DIEU,
MEMBER OF THE IMPERIAL ACADEMY OF MEDICINE,
PERPETUAL PRESIDENT OF THE MEDICAL SOCIETY OF OBSERVATION OF PARIS,
ETC., ETC., ETC.

My dear Sir,

When in days now long past—days among the happiest of my life—I made my first steps in the study of Clinical Medicine under your guidance, I had many opportunities of learning that your critical severity in estimating your own work, was not more remarkable than the indulgence with which you noticed the humblest efforts of others in the difficult path of scientific observation. Had not this trait in your character fixed itself in my memory, I should not have ventured to offer you, as I now do, a work which I know to be so unworthy of bearing your distinguished name.

Since those days the importance of minute observation and of numerical collation of facts has been so thoroughly recognized in Pathology—the justness of the principles for which you struggled is now admitted to be so obvious—that people only wonder those principles should so long have been opposed. But if this be one of your scientific triumphs, from it has originated a yet greater. There is a study, but now springing into life—that of the Science of History—and for the establishment of that science, it is commonly conceded, the Numerical Method ranks as an indispensable instrument. And it is not too much to affirm, that the successful application of numbers to medical facts—facts which, it was long held, lay *ex naturâ rerum* beyond the pale of numeration—has paved the way for its application to the vaster, if not the loftier, themes of moral and political philosophy. So true is this, that the future historian of mental civilization, in faithfully performing his task, must ofttimes mark his page with the name LOUIS.

I am, my dear Sir,
With profound respect,
Your faithful friend,
W. H. WALSHE.

LONDON, *December*, 1861.

ADVERTISEMENT TO THE THIRD EDITION.

THE present edition has been carefully revised; much new matter has been added, and the entire work in a measure remodelled.

Numerous facts and discussions, more or less completely novel, will be found in the description of the principles of physical diagnosis; but the chief additions have been made in the practical portions of the book. Several affections, of which little or no account had been given in the previous editions, are now treated of in detail. Functional disorders of the heart, the frequency of which is almost rivalled by the misery they inflict, have been closely reconsidered; more especially an attempt has been made to render their essential nature clearer, and consequently their treatment more successful, by an analysis of their dynamic elements.

While we are, on the one hand, enabled honestly to affirm that the medicinal means of controlling organic diseases of the heart have of late years improved, we can, on the other, lay fair claim to still greater advance in a hygienic point of view. And hence it comes, that the hopeless motto of Corvisart's work on cardiac diseases, "hæret lateri *lethalis* arundo," infinitely well-chosen in his time, grows yearly less and less appropriate.

CONTENTS.

PAGE

Dedication . . . v

Advertisement . . . vii

Introduction . . . 17

I. Clinical Topography of the Heart and Great Vessels . . . 17

II. Weight, Size, and Measurements of the Heart . . . 25

PART I.

CLINICAL PHYSICAL EXAMINATION OF THE HEART AND BLOODVESSELS.

§ I. The Heart . . . 29

Section I.—Inspection . . . 29

II.—Application of the Hand . . . 34

III.—Mensuration . . . 44

IV.—Percussion . . . 45

V.—Auscultation . . . 54

§ II. Great Arteries . . . 106

Section I.—Inspection . . . 106

II.—Application of the Hand . . . 107

III.—Percussion . . . 109

IV.—Auscultation . . . 110

§ III. Venous System . . . 115

Section I.—Inspection . . . 115

II.—Application of the Hand . . . 117

III.—Auscultation . . . 118

Auto-audible Murmurs . . . 124

PART II.

DISEASES OF THE HEART AND GREAT VESSELS.

PAGE
Diseases of the Heart 127

Section I.—Dynamic Diseases 127

§ I. Elementary Dynamic Changes 130
A.—Perverted Innervation 130
I.—Cardiac Hyperæsthesia 130
II.—Cardiac Paræsthesia 132
III.—Cardiac Anæsthesia 133
B.—Morbid Motor States of the Heart 133
I.—Cardiac Motor Paralysis 133
II.—Cardiac Spasm 135
III.—Cardiac Cramp 135
IV.—Perversion of Rhythm and of Force of Cardiac Contraction 136
V.—Action Perverted in Frequency 138
VI.—Action Perverted in Quickness 140
VII.—Cardiac Tremor 141

§ II. Composite Dynamic Changes, or Functional Diseases 142
I.—Simple Palpitation 142
II.—Palpitation of Irregular Rhythm and Force 146
III.—Decreased Action and Syncope 148
IV.—Angina Pectoris 156
V.—Pseudo-angina Pectoris 166

Section II.—Organic Diseases of the Heart 168

§ I.—Anæmia of the Heart 168

§ II.—Congestion 168

§ III.—Accumulation of Blood in the Heart's Cavities . 170

§ IV.—Cardiac Inflammations 171
I.—Pericarditis 171
II.—Endocarditis 197
III.—Endo-pericarditis 206
IV.—Carditis or Myo-carditis 207

§ V.—Cardiac Hemorrhages 210
I.—Intra-muscular 210
II.—Hæmo-pericardium 210
III.—Hæmo-endocardium 211

PAGE
§ VI.—Alterations of Secretion 212
I.—Œdema of the Heart 212
II.—Hydropericardium 212
III.—Pneumopericardium 214
IV.—Pneumo-hydropericardium 214
V.—Pneumo-hydropericarditis 215

§ VII.—Alterations of Nutrition 216
I.—Atrophy 216
II.—Hypertrophy 219
III.—Dropsy and Serous Flux of Cardiac Origin . . . 238
IV.—Dilatation 244
V.—Alterations of Consistence 257
A.—Softening 257
B.—Induration 259
VI.—Adventitious Products 259
Class I.—Non-plastic 259
A.—Calcification 259
B.—Fatty Precipitation 260
I.—Fatty Infiltration 260
II.—Fatty Metamorphosis 260
C. D.—Amyloid and Saccharine Precipitates . . 276

Class II.—Blastemal Formations 277
Sub-Class I.—Deposits 277
A.—Syphilitic Deposit 277
B.—Tubercle 277
Sub-Class II.—Growths 279
Carcinoma 279
Sub-Class III.—Pseudo-Tissues . . . 282
Cirrhosis 282

Class III.—Parasites 283

§ VIII.—Diseases of the Orifices and Appendages . . 284

§ IX.—Aneurisms of the Heart and Valves . . . 307

§ X.—Rupture and Perforation of the Heart . . . 310

§ XI.—Malpositions of the Heart 313

§ XII.—Intracardiac Blood-concretions 316

§ XIII.—Malformations of the Heart 323
I. Unattended with Symptoms 323
II. Attended with Symptoms—Cyanosis . . 324

§ XIV.—Injuries and Wounds of the Heart . . . 334

PAGE
DISEASES OF THE GREAT VESSELS 336
SECTION I.—AORTA 336
§ I.—SIMPLE PULSATION 336
§ II.—HYPERÆSTHESIA 341
§ III.—INFLAMMATION 341
§ IV.—ATHEROMA AND CALCIFICATION 344
§ V.—ANEURISM 345
I.—ARCH OF THE AORTA 346
II.—DESCENDING AORTA 369
III.—ABDOMINAL AORTA 375
IV.—VARICOSE ANEURISMS OF THE ARCH . . . 381
§ VI.—COARCTATION AND OBLITERATION OF THE AORTA . . 384
A.—ARCH OF THE AORTA 384
B.—THORACIC AND ABDOMINAL AORTA . . . 389
SECTION II.—CORONARY VESSELS 392
SECTION III.—PULMONARY ARTERY 393
§ I.—BLOOD-CONCRETIONS 393
§ II.—INFLAMMATION 399
§ III.—ULCERATION 401
§ IV.—RUPTURE 401
§ V.—GENERAL DILATATION 402
§ VI.—ANEURISM 402
§ VII.—ATHEROMA AND CALCIFICATION 404
§ VIII.—CANCER 404
SECTION IV.—PULMONARY VEINS 406
§ I.—INFLAMMATION 406
§ II.—DILATATION 406
§ III.—RUPTURE 407
§ IV.—CANCER 407
§ V.—BLOOD-CONCRETIONS 407
SECTION V.—DUCTUS ARTERIOSUS 408
§ I.—PATENCY IN THE ADULT 408
§ II.—ANEURISM 408
APPENDIX 409

A PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART AND GREAT VESSELS.

> . . . "Who knows but that one may discover the works performed in the several offices and shops of a man's body, by the sounds they make, and thereby discover what instrument or engine is out of order?"—R. HOOK, 1705.

INTRODUCTION.

I.—CLINICAL TOPOGRAPHY OF THE HEART AND GREAT VESSELS.

1. THE heart, seated in the lower part of the anterior mediastinum, is held *in situ* directly by the great vessels, arterial and venous, and, indirectly, through the pericardium, by the diaphragm. These attachments, fixing the base only of the heart, permit free play to its general mass, which in point of fact hangs loosely in the pericardial sac. This looseness of attachment, and this freedom of movement, essential to the physiological well-being of the organ, entail a specific inconvenience in the extreme facility with which the heart, in this respect almost rivalling the uterus, undergoes various displacements.

2. Lying obliquely (with its long axis directed forwards, downwards, and from right to left, at a slightly varying angle with the mesial plane), the base of the organ corresponds, anteriorly, to both third costal cartilages, and the apex to the sixth left costal cartilage; while, posteriorly, the upper edge of that base lies opposite the fourth, the lower opposite the seventh, or even the eighth dorsal vertebra, separated from the spine by the aorta and œsophagus. A line, carried horizontally backwards from the apex, falls in the majority of adult males on a spot seated less than two inches to the left of the body of the eighth dorsal vertebra.

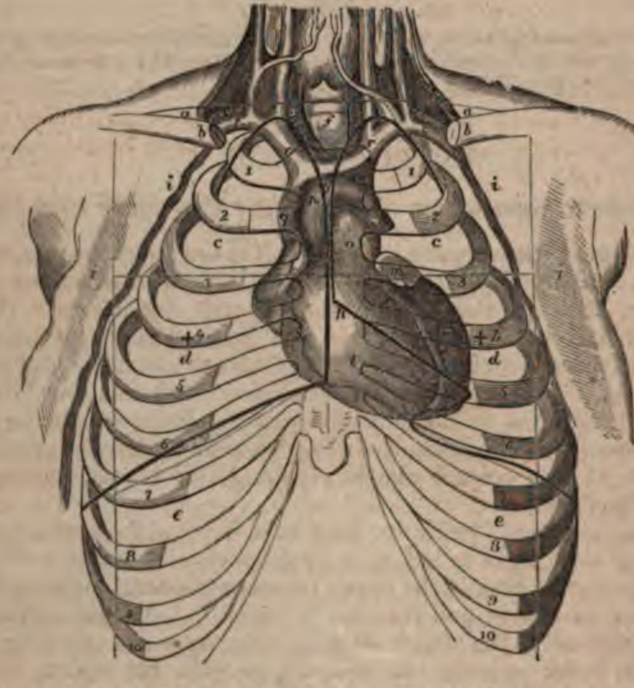
3. The postero-inferior surface of the organ, somewhat flattened, rests upon the central tendon of the diaphragm; the supero-anterior lies in apposition partly with the left and right lung, partly, between these organs and especially at its lower part, with the walls of the chest.

4. Of the two main borders of the heart, the right, notably the

thinner, lies lower, nearer the anterior chest-wall, and is habitually more horizontally placed and straighter than the left.

5. The heart occupies the entire of the lower sternal, with certain portions of the left and right mammary, regions, and inclines to encroach, more or less according to the amount of repletion of its cavities with blood, on the lower aspect of the infra-clavicular regions—an encroachment practically more frequent and significant on the right than the left side.

6. The relationship of the different compartments and orifices of the heart to the surface is a matter of which the clinical student cannot exaggerate the importance; upon correct notions concerning this relationship essentially hinges the diagnosis not only of certain diseases of the heart itself but of neighboring structures. The topographical statement I am about to submit for his guidance embodies the results of observations continued for a number of



Diagram, exhibiting the relationship of the heart and great vessels to the lungs (in moderate inspiration), and to the regions of the chest. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, integuments turned back; † †, nipples; *k*, right auricle; *l*, right ventricle; *m*, left auricle, appendix almost solely seen; *n*, left ventricle; *o*, pulmonary artery; *p*, arch of aorta; *q*, vena cava superior; *r r*, innominate veins; *s*, innominate artery; *t t*, subclavian veins. The dotted lines indicate the outlines of the thoracic regions; the dark lines, the edges of the lungs. The heart and vessels are supposed to be full.

years, and made by driving long needles into the bodies of males, at various fixed points perpendicularly to the surface, before the abdomen or thorax had been opened. The subjects, thus treated, had been cut off by all varieties of disease indiscriminately: collation of the observations made upon the whole series at once showed, as might have been anticipated, that the cases bore division into four classes—those in which the position of the organ was apparently normal; those in which intrinsic disease had perverted the relationships of its several parts; those in which extrinsic disease had affected its topography; and those in which intrinsic and extrinsic disease had both severally produced their effects. In the present place the normal state will alone be considered. The outlines and spots indicated only apply with precision to the male body examined in the recumbent posture, and free from any notable amount of physiological peculiarity either in the form of the chest, the bulk of the lungs, or the mass of the solid, and fulness of the hollow, abdominal organs. Nor must it be forgotten that the average result only is here presented—on either side of which slight oscillations, in all conceivable directions, are compatible with perfect functional health of the thoracic organs.

7. (A.) *Relationships to the anterior surface of the chest.*—The right auricle reaches, more or less extensively, according to the amount of blood stagnating in or moving through the right side of the heart, into the right mammary region—mainly on the level of the third cartilage and fourth interspace, slightly on that of the second space. In cases of extreme distension this auricle, reaching the fifth right cartilage below, may stretch upwards almost to the inferior edge of the second cartilage.

8. The right ventricle corresponds mainly to the lower sternal, and the left mammary regions. Its inferior, and nearly horizontal, border stretches from the fifth right cartilage to the point at which the apex beats in the fifth left space midway between the left edge of the sternum and a line let fall vertically from the left nipple, coasting as it passes the upper part of the sixth left cartilage. The vertical span of this ventricle at the left edge of the sternum reaches from the third to the sixth cartilage. The base of the ventricle, running slantingly from above downwards and from left to right, encroaches slightly (more inferiorly than superiorly) on the right mammary region, between the third interspace and fifth cartilage.

9. The left auricle lies deeply behind the root of the pulmonary artery—little but the appendix (Diagram, *m*) being visible anteriorly, as the organs lie *in situ*. The middle point of this auricle, from above downwards, corresponds to the cartilage of the third rib; the auricle encroaches to a variable extent on the second and third interspaces.

10. The left ventricle, reaching vertically from the third to the upper edge of the sixth left cartilage, occupies a portion of the left

mammary region. A comparatively narrow strip only of this ventricle is visible anteriorly (Diagram, a).

10*. The heart, then, as a whole, extends, vertically, from the second space to the sixth cartilage; and transversely, from a little within the left nipple to a finger's breadth or more to the right of the sternum: these are the limits of the *deep cardiac region*. The entire of the left ventricle, the greater part, by far, of the left auricle, and a large portion of the right ventricle, towards the apex, lie to the left of the sternum: and on the level of the fourth cartilage, the widths of heart-substance, lying on either side of the left border of the sternum, are very closely the same.

11. A line, carried directly backwards from the apex of the upper angle of the fourth left interspace, will fall as nearly as possible on the centre of the heart's surface.

12. The chief relationships of the lungs to the heart, applicable in clinical study, are as follow. The entire of the right auricle, and about one-third of the right ventricle, are covered by the right lung, which passes vertically downwards, coasting the middle line, to the lower edge of the heart: the entire of the left auricle, the upper left part of the right ventricle, and the entire of the left ventricle, except a variable, but small, portion towards the apex, are covered by the left lung. The portion of heart uncovered by lung, thus belonging almost exclusively to the right ventricle, is of rudely triangular shape. The upper angle of the triangle corresponds to the middle line on the level of the fourth cartilage, where the anterior edge of the left lung diverges from its fellow; while the base is formed by that portion of the lower edge of the heart lying between the middle line and the spot at which the apex beats: this is the *superficial cardiac region*. The vertical side of the triangle measures on an average, in the middle-sized adult male, two inches—the horizontal, two inches and a half—the oblique, nearly three inches. (*Vide* Diagram.) But, although it be certain that the triangular outline is of sufficiently constant occurrence to be regarded as the typical condition, it must be admitted the form varies, sometimes resembling a parallelogram, sometimes nearly square, sometimes wholly irregular.

13. *Valves.*—(*a.*) The upper or free edge of the *pulmonary* valves lies horizontally, and in the mass of persons a shade above the upper edge of the third left cartilage, the body of the valve consequently a little lower than this—the left edge of the sternum having closely the same width of the vessel on both sides. Luschka[1] places this valve somewhat higher; but, in point of fact, almost all the spots of importance are fixed by him at a slightly higher level of the chest than would be justified by the average result of my observations.

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[1] Die Brustorgane des Menschen, p. 12—a work of most conscientiously elaborated detail.

(*b*.) The *aortic* valves, lying horizontally a very little further inwards and lower than the pulmonary, correspond to the union of the sternum and third left cartilage.

(*c*.) The attached edge of the *tricuspid* valve, slantingly placed across the sternum from above downwards, and from left to right, inclines from the neighborhood of the sternal edge of the third left interspace nearly to the sternal end of the fourth right interspace or fifth right cartilage.

(*d*.) The attached edge of the *mitral* valve lies almost horizontally about one-quarter of an inch lower than the attached bases of the aortic valves, very slightly further inwards than these, and deeper than the tricuspid valve. The attached border lies on the level of the union of the third cartilage with the sternum, nearer, as a rule, the upper than the lower border of this: I have, however, found the body of the valve as low as the mid-height of the third interspace.

14. The transverse distance between the nearest points of the right pair of valves, tricuspid and pulmonary, averages half an inch; the left sets, aortic and mitral, fall within the same vertical plane.

15. A superficial area of half an inch square will include a portion of all the four sets of valves *in situ;* an area of about one-quarter of an inch, a portion of all except the tricuspid. The isolation and distinction of the sounds and murmurs produced at the several orifices, if possible at all, must be effected on some other principle than that of direct conduction to the nearest spot of the chest-surface—that spot is in point of fact practically the same for all.

16. The aorta, rising from the sternal end of the third left cartilage, nearly opposite the union of the two sternal regions, ascends at once into the upper sternal region, at first under cover of the pulmonary artery; and, inclining to the right, reaches by its right border the inner and upper part of the second right costal cartilage, occasionally touching on the inner edge of the first interspace: thence crossing, almost horizontally, the upper sternal region, on the level of the first interspace, and in front of the trachea just above its bifurcation, it passes backwards and downwards to the left side of the body of the third dorsal vertebra. Sometimes the entire of the arch lies a little higher than this.

17. The pulmonary artery ascends from the position of its valves, with slight inclination to the left side, as far as the second left cartilage or first interspace, its point of bifurcation; in this short course it passes somewhat backwards, and is consequently further somewhat from the surface of the chest opposite the second, than opposite the third, cartilage. It occupies a portion of the upper sternal region, encroaching on the edge of the left infra-clavicular.

18. The arteria innominata rising on the level of the upper edge of the first interspace, or on that of the lower part of the first cartilage, behind the right half of the upper sternal region, passes up-

wards and to the right, bifurcating to the right of the trachea, and behind, or a little above, and to the right of, the sterno-clavicular joint: in its course it lies partly in front of the right half of the trachea.

19. Inasmuch as the sounds and murmurs produced in or transmitted along the arch of the aorta, the pulmonary and the innominate arteries are, as a rule, severally best isolated at the second right, the second left, and the first right costal cartilages—these cartilages may for clinical purposes be respectively called the aortic, pulmonary, and innominate. An important qualification to this general truth must, however, be added. Inasmuch as the pulmonary artery at its root lies in front of, and nearer the surface than, the aorta, with which it stands in close union, it follows that at the actual base the vibrations of the aortic valves are not only conveyed to the surface through the pulmonary artery, but having reached the walls of this vessel may be freely transmitted along them, and hence that either sound or murmur really produced at the mouth of the aorta may be better audible at the cartilage we have just named pulmonary, namely, the second left, than at the second right or aortic.

20. The superior vena cava lies along the right edge of the ascending portion of the arch of the aorta, near the right border of the sternum, from the first interspace to the third cartilage; in the former spot occurs the union of the two innominate veins.

21. The pericardial sac (representing a cone of which the base, lying inferiorly, is fixed to, and partly incorporated with, the upper surface of the central tendon and muscular portion of the left ala of the diaphragm, while the apex, placed superiorly, embraces the lower two inches of the great vessels) extends from the second to the seventh left costal cartilage. In rare instances, however, where the pericardium proves wholly free both from present disease and from anatomical evidences of past morbid distension, the cavity of the sac reaches the level of the first costal cartilage.[1]

22. A larger portion of the pericardial sac lies to the left than the right of the middle line—the superficial areæ of the two portions standing to each other very closely in the ratio of 3 : 2. Hence the extent of reflected pleura is greater on the left than the right side—a fact wherefrom some diagnostic inferences indirectly flow.

23. The sac of the pericardium lies further from the anterior chest-wall superiorly than inferiorly—a mode of relationship which lends its aid in rendering the detection of fluid less early, as a rule, at the upper than the lower part of the cavity.

24. The action of the heart is accompanied, as vivisections prove, by alterations in form, size, axis, and position; in systole, in diastole, and in repose the organ is specially characterized in regard of

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[1] H. Hodson, U. C. H., Males, vol. ix. p. 20.

these four attributes. But to what extent are these changes, produced by the heart's own working, clinically appreciable? In the perfectly natural state not one of them can be substantiated except the visible and palpable alternate advance of the heart's apex to, and recedence from, the surface during systole and diastole. But in certain morbid states it may be ascertained, by the help of the eye and hand combined, that during the systole the organ twists spirally on its longitudinal axis from right to left, especially towards the apex, which at the same time comes forward; while the converse movements attend the diastole. In certain cases, too, where obstruction to the intra-cardiac circulation is carried to a high point, alterations in size and even in shape may be established by careful percussion.

25. It has been already signified [6] that the precise position of the heart within the thorax varies independently of disease either in, or beyond, the organ itself. This variation expressly affects height—the organ lies a little lower or a little higher than natural.

26. Age exercises an influence in this direction. In early youth the organ lies higher than in the mass of persons advanced in life. Many conditions probably combine to lower its position as years roll on: the increasing weight and bulk of the heart itself, a normal effect of prolongation of life, drag it down; the pressure in aged people of emphysematous distension of the lungs pushes it down; and the aorta, atheromatous or actually calcified in old age and hence impaired in elasticity, sustains the weight of the organ less effectively than in youth.

27. The heart of the civilized adult female, of the present day, lies on the average slightly higher than that of the adult male. I am inclined to believe this difference the result of dress: the habit of tight lacing in the female will tend to lower or raise the heart according as the line of maximum pressure is low or high—consequently, as the fashion of low waists prevails at the present day, to raise it. The tight abdominal belt worn by some males exercises a similar, though less marked, influence.

28. Alteration of posture of the body affects the site of the organ also. The heart falls downwards somewhat (if its substance be weighty, the fall may equal an inch) in the erect position, and comes more forward than in decumbency. Changing the position in decumbency from the right to the left side will carry the heart an inch, or even more, to the right or left of the position it occupies when the individual lies on the back. Now this is a fact of considerable importance, as it shows that, especially when such change is only moderate in amount, lateral movableness of the dull percussion-sound of the deep cardiac region cannot be accepted as conclusive of the existence of fluid in the pericardium. In the prone posture and in decumbency on the left side the heart comes more fully towards the chest-wall than in the supine posture, or in

decumbency on the right side—facts which have their clinical application in the diagnosis of pericardial friction-sound.

29. The mode of dissolution exercises an influence on the precise position of the heart *post-mortem*. If the immediate mechanism of death have been asphyxial the heart will lie lower than if it had been syncopal, and the amount of the difference will be in the direct ratio of the degree to which the asphyxial or syncopal phenomena have been severally carried. In comparing the position of the heart after death with that supposed to have been established during life, the mechanism of the final struggle must consequently be borne in mind.

30. Locomotion of the heart attends each act of respiration. Inspiration, by carrying the diaphragm downwards, lowers the heart, sometimes by an entire interspace; and, by bringing a thick stratum of the left lung in front of the organ, removes it somewhat from the thoracic walls: the weakening effect thus produced on the heart's impulse and sounds is in healthy persons very perceptible. The position of the valves and the maximum points of the cardiac sounds are proportionably lowered; but it is to be remembered, that the depression of the diaphragm displaces the base more than the apex of the heart. This depression tends to lengthen momentarily the great vessels, especially the ascending portion of the arch of the aorta. The converse effects are produced by expiration. The amount of this locomotion is unequal in the two sexes. Inasmuch as the calm breathing movement of the male is essentially abdominal and inferior costal (phrenic), while that of the female plays specially on the upper third of the chest, the heart changes its place more extensively with the respiratory act in the former than in the latter sex.

31. (B.) *Relationships to the posterior surface of the chest.*—On the whole, these are of much less clinical importance than the anterior group.

32. Of the heart itself the parts which come nearest the posterior chest-wall are the left auricle essentially, and the left ventricle to a small extent at its upper part. These, the portions of the heart lying furthest backwards, are separated from the surface by the pericardium, superiorly and transversely by the left bronchus, vertically by the œsophagus, the thoracic aorta and the vertebral column—on the left side by the lung, while on the right (scarcely more than one quarter of the left auricle lies to the right of the mesial plane of the body) the lung is separated from the heart proper by the right pulmonary veins, vena cava inferior, and right main bronchi. As, anteriorly, scarcely any of the left auricle is visible on the surface, so, posteriorly, a very small portion only of the lower edge of the right auricle nears the surface.[1]

33. The descending portion of the arch of the aorta, continued

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[1] Luschka, loc. cit., Taf. iv.

into the thoracic aorta, passes on the left of the spinal column behind the left pulmonary artery, the left main bronchus, the left auricle, and the posterior base of the left ventricle. Murmurs produced in the course of the thoracic aorta are well audible in the left vertebral groove from the fourth to the eleventh ribs; those produced at the aortic valves and conducted in the walls of and within the vessel, are, as far as the back is concerned, best heard in the left vertebral groove from the third to the fifth spaces—assuming that the conducting faculty of the lungs and other adjoining structures has undergone no deviation from the standard of health. Aortic or pulmonary murmurs are with difficulty transmitted in a direct line through the intervening tissues backwards to the posterior surface of the chest.

34. The mitral orifice corresponds most nearly to the level of the sixth rib and space in the left vertebral groove—and here the murmurs produced at that orifice are best audible by direct conduction backwards.

II.—WEIGHT, SIZE, AND MEASUREMENT OF THE HEART.

A.—In Health.

35. Inasmuch as the heart varies very sensibly in bulk with the stature and mass of the whole body, and also with the age of the individual, it follows that a general standard applicable with absolute precision to all persons, even of the same sex, is not attainable. For practical purposes, it may, however, be assumed that nine and a half ounces represents the average weight of the healthy male heart, eight and a half ounces that of the female organ, between the ages of thirty and sixty. A material deviation in either direction from these estimates would indicate a failure or an excess of nutrition.[1]*

36. The discoveries, independently made by Clendinning and Bizot, that the weight of the heart, the thickness of its walls, and the length and breadth of its mass, increase, as age advances, have their clinical application, especially in individuals of the male sex, in whom the change is much more sensible, both in amount and in steadiness, than in females. A weight over ten ounces in a male, aged five-and-twenty, would certainly render the existence of morbidly hypertrophous growth more than probable, whereas at the age of fifty a weight of ten ounces and a half might have coincided with a perfectly healthy state of the organ in all its local attributes and systemic influences. It must, however, be confessed that in the present state of knowledge there are intermediate degrees of over-weight, the healthy or morbid nature and tendencies of which can only be determined by reference to the past functional condition of the organ: in other words it results positively from my

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[1] These estimates not only closely represent the mean, but also the most common, weights in a large number of cases.

observations that a given weight of heart, in a given sex, at a given quinquennial period of life, with a given stature, and a given weight of the whole body, will in one instance have belonged to an individual whose heart has never caused him the least trouble, in another to a patient who has suffered from many of the minor, perhaps some of the graver, evils of hypertrophy. Forced by experience to accept this conclusion, I attach little importance to slight excess (from half an ounce to an ounce and a half) above the alleged normal par of weight.

37. The thickness of wall of the different parts of the heart has been most carefully established by M. Bizot,[1] and from his tables the following excerpt, having real practical significance, is arranged. It displays the mean thickness in French lines of the walls of the ventricles and septum, exclusive of the columnæ carneæ, between the ages of sixteen and eighty:—

	MALES.			FEMALES.		
	Base.	Middle.	Apex.	Base.	Middle.	Apex.
Left ventricle . .	$4\frac{68}{122}$	$5\frac{19}{122}$	$3\frac{95}{122}$	$4\frac{3}{8}$	$4\frac{4}{5}$	$3\frac{13}{30}$
Septum		$4\frac{5}{12}$			$4\frac{1}{36}$	
Right ventricle .	$1\frac{112}{122}$	$1\frac{99}{244}$	$1\frac{2}{61}$	$1\frac{2}{3}$	$1\frac{7}{24}$	$0\frac{673}{720}$

Hence it appears, the middle part of the left and the base of the right ventricle are respectively the thickest parts. From other tables it follows that, whereas the thickness of the left ventricle steadily increases with advancing years from puberty to extreme old age, that of the right ventricle remains almost stationary. The earlier the period of life, the less the difference between the thickness of the walls of the two ventricles.

38. The mean thickness of the wall of the right auricle has been estimated at about 1 line Fr. by M. Bouillaud—that of the left $1\frac{1}{2}$ line.

39. The absolute and relative capacities of the four compartments of the heart, during life, cannot be estimated with precision from examination of their areæ after death. The manner of dissolution must obviously affect to a serious degree the relative condition of the four cavities; and hence it is that the results of different observers vary so widely as to render them all untrustworthy. As to absolute capacity, nothing can be stated with even an approach to certainty. The capacity of the two ventricles is probably equal —that of the right auricle slightly greater than that of the left, say in the proportion, taught by M. Cruveilhier, of 5 : 4.

40. The establishment of the width of the four orifices of the heart, though by no means so easy as might on first thought be imagined, is not beset with the extreme difficulties attending that of the capacity of its cavities. The simplest and most correct plan consists in laying a wet piece of thin cord along the attached border

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[1] Mém. Méd. Soc. d'Observation de Paris, t. i. p. 280.

of the auriculo-ventricular, and the free border of the arterial, valves: there is no necessity to stretch the parts, as the cord follows closely the inequalities of the surfaces.[1] The mean measurements of M. Bizot, from the ages of sixteen to eighty, are subjoined in French lines:—

	Auriculo-Ventricular Orifices.		Arterial Orifices.	
	Left.		Left.	
	Males.	Females.	Males.	Females.
Mean . .	45$\frac{17}{30}$	41$\frac{1}{3}$	31$\frac{15}{61}$	28$\frac{2}{3}$
	Right.		Right.	
Mean . .	54$\frac{18}{61}$	48$\frac{1}{4}$	32$\frac{28}{61}$	30$\frac{7}{60}$

Hence it appears that the mean excess in width of the right over the left auriculo-ventricular orifice equals about 8½ lines in males, 6$\frac{11}{12}$ lines in females: the pulmonary orifice exceeds the aortic in width by a mean amount in males of 1$\frac{1}{10}$ line, in females of 1¼ line. Hence, while the excess of width on the right side of the heart is relatively greater in the male at the auriculo-ventricular, it is relatively greater in the female at the arterial, orifice. In persons who have passed the age of fifty it would appear from M. Bizot's figures that the aortic orifice is wider than the pulmonary by a mean amount in males of 1 line—in females of $\frac{1}{18}$ of a line. This deviation from the rule of earlier years is plausibly referred to the greater frequency of atheroma and calcification in the artery of the left side, whereby the resistant power of the vessel being diminished it gradually undergoes dilatation.

41. As in the case of the right and left compartments of the heart, so in that of their orifices, it has been suggested that the difference existing at death is directly connected with and depending upon the manner of dissolution—and either does not exist at all, or exists only to a slight degree, during life. This appears to me less unlikely to be true—or true to the full degree intended—of the auriculo-ventricular than of the arterial orifices.

B.—In Disease.

42. The diseases of the heart itself and its membranes of course variously affect, and indeed in some cases are mainly signified by changes in, the weights and measurements we have just been referring to. But there are certain diathetic affections in which the bulk and measurements of the heart undergo very sensible alteration, as a consequence probably of the lessening mass and failing nutritiveness of the blood. To these conditions must, probably, be referred the deficiency in the mean weight and size of the heart of persons cut off by chronic phthisis and cancer, as demonstrated by MM. Louis and Bizot. It may be that a phthisical individual, whose

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[1] "What to Observe at the Bedside." By the Lond. Med. Soc. of Obs., p. 109, Ed. 2.

heart only reaches the par of health for his age and sex, is nevertheless clinically the subject of hypertrophy: his amount of heart is beyond the legitimate wants of his impoverished system.

43. On the irritant qualities of the circulating fluid in Bright's disease probably depends the excess of bulk of the heart in the majority of individuals suffering under that complaint—and who, while free from valvular affection, are not, symptomatically considered, the subjects of hypertrophy.

PART I.

CLINICAL PHYSICAL EXAMINATION OF THE HEART AND BLOODVESSELS.

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§ I.—THE HEART.

44. THE physical methods of examination applicable to the heart are: Inspection; Application of the hand and Palpation; Mensuration; Percussion and Auscultation.

SECTION I.—INSPECTION.

45. Inspection directs itself to: the form of the cardiac region; the condition of its integuments; and the visible impulse of the heart.

A.—IN HEALTH.

46. (*a.*) *Form.*—In perfectly normal chests the part of the walls lying to the left of the middle line, and corresponding to the heart, does not differ perceptibly in form from that placed to its right; these two divisions of the thorax are symmetrical. But individuals, who have never suffered from pulmonary or cardiac disease, occasionally present a moderate excess of convexity of the cardiac region generally, as a result of natural conformation, of curvature of the spine, or of change of form produced by influences of a non-morbid kind. So, too, physiological depression, or flattening, of the lower part of the præcordial region, in particular, sometimes occurs. M. Woillez states that such depression, when really non-morbid, is observed in the corresponding right region also—is in fact symmetrical; but, I confess, I cannot wholly agree with him on this point. Without questioning the general accuracy of his statement, I must observe that I have occasionally seen depression limited to the left infra-mammary region, both in the male and female, where the most careful examination failed to elicit evidence of present or past disease, either of the heart, liver, lung, pleura, or diaphragm.

47. (*b.*) *Integuments.*—The præcordial interspaces are of the same width, and lie on the same plane as their fellows on the opposite side; and the soft parts have the same characters on both.

48. (*c.*) *Impulse.*—In the majority of healthy persons the impulse is visible only at the apex, which beats in the fifth interspace, and somewhat against the sixth rib, about midway between the line of the nipple and the left border of the sternum; the area of visible impulse does not exceed a square inch. Various physiological acts modify the precise spot of visible impulse. Thus changes of posture elevate, depress, throw it upwards or backwards: inspiration lowers it somewhat, and by carrying the lung in front of the heart weakens its force; expiration has the converse effects. A full meal or flatulent distension of the abdomen raises the apex-beat somewhat, and throws it to the left; pregnancy has the same effect. As a rule, thin, tall persons have an impulse of greater visible extent than the short and stout: in the obese, none can be detected; in persons with short sternum, it can be seen in the epigastrium. Habitually, it is more extensive in males than in females, and in persons of nervous than of other temperaments.

To the eye the impulse seems gently heaving and gliding from above downwards and from right to left, of brief duration, free from abruptness, regular in rhythm, and single.

B.—IN DISEASE.

49. (*a.*) *Form.*—The cardiac, or præcordial, region becomes arched forwards in the course of pericardial effusion. I have never observed this change of form in pericarditis previous to the occurrence of liquid effusion; but it may occur while the fluid is yet very small in quantity. The intercostal spaces widen—their muscular planes eventually almost protruding beyond the level of the ribs; while the left border of the sternum is pushed more or less, never more than slightly, forwards: change of form so marked as this indicates abundant effusion, and may reach from the sixth to the second left cartilages inclusive. Hypertrophy of the heart, especially of the left side, increases similarly the convexity of the cardiac region from the third to the seventh cartilages, and widens, but does not produce actual bulging of, the interspaces: pericardial adhesions, especially if associated with agglutination of the pericardium to the sternum, increase the prominence caused by any given amount of coexistent hypertrophy. Solid accumulations in the lower part of the anterior mediastinum likewise arch the superjacent walls, and have occasionally caused great obscurity in diagnosis.

50. On the other hand, depression or excavation of the præcordial region may occur during the absorption-period of pericarditic effusion: commonly at the lower part of the region; occasionally, as I have once seen,[1] above its natural limits. In this instance the excavation formed during convalescence at the first and second left interspaces, close to the sternum. It has been the habit, on speculative grounds, to ascribe any such depression following pericarditis

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[1] Clinical Lecture, case of Craddock, "Lancet," vol. i. p. 144, 1849.

to the influence of adjacent pleurisy; but in point of fact, the change of shape sometimes occurs where all physical signs have argued against the existence of the latter inflammation.

51. (*b.*) *Integuments.*—The integuments of the cardiac region are sometimes markedly more œdematous than those of other parts of the chest, in cases of pericardial effusion lapsing into the chronic state. The phenomenon, in itself unimportant, derives interest from its occasionally giving rise to sounds, audible with the stethoscope, and simulating moist rhonchi and friction-sound.

52. (*c.*) *Impulse.*—Disease alters the position, extent, force, character, and rhythm of the heart's visible impulse.

53. The apex-beat is in the first place changed in position by a variety of diseases of the lungs, pleura, mediastinum, and abdominal organs;[1] but affections of the heart itself, and of its external covering likewise displace it. All enlargements of the heart depress the apex-beat, and may carry it as low as the seventh interspace, or eighth rib: if both sides of the organ be equally affected, that point is commonly displaced to the left; if the right side be the main sufferer, the impulse is chiefly visible to the right, behind and below the sternum; where the left side is alone, or chiefly, diseased, the apex may be seen at variable distances from the natural spot, to about three inches and a half to the left of the nipple.[2] If, as is not very uncommon, hypertrophy affect, almost exclusively, the part of the left ventricle adjoining the mitral orifice, the base of the organ falls disproportionately to the rest, and the apex is thrown forwards. Procidentia of the apex in general hypertrophy is, *cæteris paribus*, greatest, where there is agglutination of the pericardial surfaces also. Aneurism of the arch of the aorta, or of the pulmonary artery, likewise lowers mechanically the point in question. It has been affirmed that atonic relaxation of the great vessels occurs in various acute adynamic diseases to sufficient amount to allow of an appreciable fall in the heart's level: I have lately observed this in a case of typhoid fever.

54. On the other hand, diminution of the size of the cavities, consequent on sudden enormous loss of blood, acting in conjunction with retraction of the great arterial trunks, slightly raises the apex-beat. It seems possible that a rude estimate of the degree of hemorrhage might be formed through this changed position of the heart—but of course only in individuals the precise point of whose normal beat was previously well known.

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[1] Enlargement of the right, as well as of the left, lobe of the liver, may displace the heart's point upwards and outwards, and sometimes give useful and unexpected aid in diagnosis. Such displacement contributed much to distinguish acute abscess with enlargement of the liver from abscess of the abdominal walls, in a case which I published some time since. (Case of Fairbanks, Clin. Lect., "Lancet," loc. cit.)

[2] The furthest point I remember to have seen is 3¾ inches outside the nipple (*vide* case of Hope, Clin. Lect., "Lancet," loc. cit., p. 415); this, too, is only possible in broad-chested persons.

But the affection which most notably raises the apex-beat is pericardial effusion. When considerable accumulation exists in a pericardium free from old or recent adhesions, if the heart be not enlarged, and if there be no adjacent pleuritic adhesions, the increase of fluid pushes the base of the heart and the great vessels backwards and upwards, and twists the apex outwards and upwards, so that it beats opposite the fourth interspace or rib directly behind, or a little outside, the nipple. The progress of elevation may be traced from day to day, as of the subsequent fall during the course of absorption. When the spot of the apex-beat is thus raised, it may always be *seen*, unless where general undulatory impulse tends to throw it into the shade: but it may or may not be *felt;* if not, the amount of effusion is in all probability very considerable. The possible sources of fallacy in interpreting this sign are fully considered with the subject of pericarditis. If during convalescence the apex gradually fall to below its natural site, hypertrophy, as an immediate sequela of the pericarditis, may be diagnosticated; for had the enlargement preceded the serous effusion, the apex would not have been raised.

I have further distinctly ascertained that where the pericardial surfaces are agglutinated together by recent and abundant exudation matter, the interstitial contraction of this will sometimes suffice, unaided by fluid in the sac, to raise the apex-beat an intercostal space.[1]

55. In the natural state of things the maximum of the heart's visible impulse corresponds to the apex of the organ; under peculiar circumstances that maximum may be transferred to the base. Thus in a case of highly-developed hypertrophy and dilatation of the left ventricle,[2] where numerous circumstances pointed to the probable existence of an aneurism of the thoracic aorta behind the base of the heart, the impulse was very notably greater about the third than the sixth interspace. As the case did not terminate fatally in the hospital, I had no means of positively ascertaining whether aneurism existed or not; but I do not believe that any affection of the heart's substance alone (except, perhaps, true saccular aneurism of the left ventricle), will thus transfer the maximum amount of visible impulse from the apex to the base. I refer here to purely systolic impulse.

56. Instead of a forward movement accompanying the systole, a sinking inwards is sometimes to be seen. In some thin-chested people, with moderate hypertrophy, while the fifth interspace rises, the fourth sinks with the systole; the organ appears to go through a see-saw movement, and produces a momentary tendency to a vacuum at the fourth. So, too, the upper part of the epigastrium sinks in sometimes with the systole—a sign originally deemed

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[1] Campion, U. C. H., Females, vol. vi. p. 39, 1852.

[2] F. Groove, U. C. H., Males, vol. viii. p. 13, April 25, 1849.

diagnostic of pericardial agglutination, and certainly, though more frequently absent, sometimes attending this state. I have known this systolic depression extend to the lower part of the sternum, and the two or three adjacent cartilages, in hypertrophy with agglutinated pericardium. Hypertrophy with dilatation will produce epigastric depression during the systole, in persons with short sternums, independently of adhesions.

In some rare cases, the visible impulse appears horizontally double and pendulum-like.[1]

57. The other conditions of impulse, except the so-called undulatory variety, are better appreciated by the hand than by inspection.

58. Undulatory impulse, suggesting the wavy motion of fluid, very variable in extent, may reach vertically from the first to the sixth interspace, and transversely from the left nipple to an inch to the right of the sternum. Its position may be altered, in the same directions as the heart itself, by changing the patient's posture. Commonly the axis of undulation is diagonal, from below upwards, and from left to right: undulation may be well marked in one interspace, imperfect or null in an adjoining one; in this case the line of current is generally horizontal.

When so fully developed that there can be no mistake about the fact, undulatory impulse is a very distinctive sign of fluid in the pericardium. But the sign unfortunately exists in a small proportion only of cases of hydropericarditis; and I do not remember to have ever satisfactorily observed it in mere dropsical hydropericardium; and a kind of pseudo-undulation is not an uncommon character of the impulse of weak and fatty, dilated hearts, especially where any physical conditions of the lung or pleura, combining with the dilated enlargement of the organ itself, tend to bring an abnormal extent of its anterior surface in contact with the chest-walls.

59. Furthermore, a variety of undulatory impulse exists in cases of pericardial agglutination, where this is combined with similar close adhesions of the præcordial pleural surfaces. Dilated hypertrophy being also present both systole and diastole are impulsive;—dimpling inwards occurs with the systole at the apex, while a space higher a slight bulging may take place at the same instant: during diastole the converse conditions occur; and these alternating actions give a very distinct wave-like movement over the surface concerned.

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[1] "Systolic action of heart, unnaturally visible in fifth interspace, from a finger's breadth to left of sternum to two fingers' breadth to left of nipple, or about three inches; movement distinctly pendulum-like—a first blow appearing to be given at the outer part of space mentioned, a second at the inner; but by application of the finger it is found that the real impulse is only at the site of the external visible impulse. When the outer point is struck by heart's apex, the inner point sinks in; when the apex recedes, the inner point recovers its natural position." Dunn, U. C. H., Males, vol. vii. p. 76; Feb. 17, 1852. The case was one of cancer of the stomach with great emaciation, no cardiac disease.

These actions may be more or less distinctly perceptible in four or even five spaces.[1]

SECTION II.—APPLICATION OF THE HAND.

60. By application of the hand, the heart's impulse, the movements of the præcordial ribs and interspaces, and the state of vocal fremitus over the cardiac region are examined.

A.—IN HEALTH.

61. The visible movement corresponding to the heart's point is felt to possess a certain amount of impulsive force; which depends on the shock of the part immediately adjoining the apex of the organ against the side.

62. The essential physiological facts of clinical import, connected with this shock, are: (*a*), its production by both ventricles close to the point of the heart; (*b*), its synchronism with the systole of the ventricles; (*c*), its synchronism with the commencement of the first sound of the heart; (*d*), its anticipation by a moment of the diastole of the neighboring arteries.

63. (*a*.) That the right ventricle plays any part in the impulse has been denied by M. Filhos, on the ground that the fibres of that ventricle, not being spirally arranged, are incapable of undergoing any other movement than those of contraction and dilatation. But, in the first place, there is no absolute certainty that the impulse depends on the action of the heart's spiral fibres at all; in the second, the phenomena of dilated hypertrophy of the right ventricle (which hypertrophy cannot be imagined to give a new direction to fibres) prove that powerful impulse may depend on that portion of the heart solely; and, in the third place, Reid, among others, has distinctly shown that the right ventricle contains spiral fibres as well as the left.

64. (*b*.) The systolic synchronism of the impulse, as commonly accredited, is not universally received. Taking the systolic coincidence as one theory, the total number amounts to four, the additional opinions standing as follow: first, the normal impulse synchronizes with the diastole of the ventricles, and this alone (Descartes, Burdach, Beau, Cartwright, and others); secondly, the normal impulse is constantly double, systolic and diastolic (Verneuil,[2] Bellingham); thirdly, a diastolic and a systolic impulse both exist, but "succeed each other so rapidly in the normal state that they become, as it were, confounded together, and cannot be distinguished from each other" (Aran).[3]

The majority of persons advocating these opinions appear to me to have embroiled this question, of which the clinical importance cannot easily be exaggerated, by mixing up with it the problem of the mechanism of the impulse. Assuming that a certain agency is the efficient cause of the shock, they infer that shock must be systolic, diastolic, or both, because the agency in question comes into force at one or other or both those periods of the heart's revolution. This is simply explaining a dubious point by one yet more dubious —*ignotum per ignotius.*

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[1] Dell, U. C. H., Males, vol. xvii. p. 148.

[2] Locomotion du Cœur, Thèses de Paris, 1852.

[3] Traduction de Skoda, p. 218, 1854.

Yet, on the other hand, clinical determination of the question is excessively difficult in the human subject; and the statical relationships of the contents of the thorax, as well as the conditions under which those contents normally move, are so gravely altered by taking away the wall of the chest, that the results of vivisections, even of large vertebrate animals, cannot be implicitly confided in. Although not unopen to objection, the observations made in cases of ectopia cordis in the human subject seem to supply the best materials for determining the time of the healthy impulse. Now Cruveilhier,[1] Skoda,[2] and Mitchell,[3] who have each carefully observed a case of this kind, though they differ in many points, agree as to the synchronism of the impulse with the ventricular systole. And this result is satisfactorily confirmed by an observation made by Bamberger on a man, aged thirty, with a clean, gaping, penetrating chest-wound, below and somewhat anterior to the nipple at the lower edge of the fifth left rib. The finger, introduced perpendicularly to the surface, encountered the smooth slippery apex: with every systole, the apex, hardened and brought somewhat to a point, glided on the finger, along the chest wall, in a direction from above downwards, and somewhat towards the left, passing down slightly beyond the inferior limit of the wound; whilst in diastole it was retracted upwards and became imperceptible.[4] I accept, then, this synchronism as a demonstrated truth—especially as the mass of observation of other kinds (*exempli gratiâ* that of the coincidence of the heart's first sound with the impulse), as likewise the weight of argument, deposes in its favor. That there is locomotion of the ventricles during their diastole, as a consequence of the influx of blood from the auricles, may be conceded; but there is a total want of evidence that in the healthy state such locomotion is productive of parietal impulse.

65. (*c. d.*) The relationship in point of time of the ventricular systole to the first sound of the heart, and to the diastole of the neighboring arteries is considered with the account of the mechanism of the heart's sounds.

66. *Clinical characters of the impulse.*—The impulse, slight in amount and imperceptible to the individual himself, mixed impulsive and gliding in character, free from abruptness or sharpness, yet decisive in rhythm, of brief duration, varies in force directly as the

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[1] Gazette Méd. de Paris, Août, 1841.

[2] Dub. Journal Med. Science, 1844.

[3] Perkussion, &c., 4e. Auflage.

[4] Quoted by Luschka, loc. cit.

muscularity of the heart, and the energy and rapidity with which its fibres contract. However, in broad-chested and stout persons, though provided with powerful hearts, the impulse may be scarcely perceptible to the hand: it may then be detected by placing a stethoscope in the fifth interspace, and applying the ear as if for auscultation. In thin, tall persons, the impulse is disproportionably strong—so also in women and children, in whom, too, it is distinguished by a certain abruptness and shortness.

Though, of course, essentially the same in position as the visible impulse, the shock may be felt a little lower than seen, namely, behind the sixth rib. This peculiarity is, however, not observable by any means in all people; it probably depends on an unusual amount of descent of the heart during systole.

67. *Modifying influences.*—Forced expiration widens the area, and increases the apparent force of the impulse; inspiration affects both conversely. The varying condition of the neighboring edges of the lungs at the two periods of each respiration fully explains this. Various acts quickening the heart's action intensify the shock—muscular exertion, walking at a rapid pace, and *à fortiori*, running, going against the wind, ascending heights, &c., have all this effect. Voluntary acceleration of the respiration will, especially in peculiar constitutions, act (indirectly, by quickening the circulation) similarly on the impulse; so too will the slight erethism of the circulation that accompanies digestion. But of all causes which act in this manner, independently of disease, moral influences and the passions are the most powerful; and among these, fear, perhaps, holds the highest place. The shock, under the agony of fear, becomes so powerful, as not only to be distinctly perceptible to the individual, but to be actually and sharply painful. Simple "nervousness" acts, to a slight degree, similarly.

68. *Mechanism of the Impulse.*—It does not appear that, if the mechanism of the healthy impulse be considered in itself alone, and without reference to the period of the heart's revolution at which the shock is effected, any one rather than the rest of the numerous theories, adduced in its explanation, involves consequences of clinical significance. In various states of disease where the manner of the impulse might, if thoroughly understood, carry with it diagnostic weight, the mechanism indubitably differs from the normal type. It would consequently be unwise to devote any lengthened space in a purely clinical work to a discussion of the multitudinous speculations on the subject; a brief statement of the doctrine, apparently the most trustworthy, seems alone required.[1]

69. The mechanism is complex. (1.) During systole the ventricles shorten, and at the same time bulge in all directions, the bulg-

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[1] As the "Segner's water-wheel," or recoil theory, ascribed to Gutbrod, and adopted with certain modifications by Skoda, has had many partisans, I think it right to observe that the combined objections of Valentin, Messerschmid, Kiwisch, and Aran seem to me to refute it victoriously.

ing being most manifest a little above the apex. This bulging leads to centrifugal peripheric pressure in all directions—among these towards the anterior wall of the chest: herein lies one component element of the impulse.[1] But only one element—it is perfectly obvious that were this, as some persons maintain, the sole cause of the shock, a sensation of movement from below upwards would attend it, and not one, as is really the case, of movement from above downwards.

(2.) A swinging movement from right to left accompanies the systole. This can be seen distinctly, nay, even felt, in some thin-chested people; its reality was besides substantiated by Bamberger in the case of penetrating chest-wound already referred to. This movement is probably a resultant of the action of the spiral fibres of the ventricles, and is indubitably forcible enough to impress the chest-wall.

(3.) But there is yet another systolic movement, that of the total heart from above downwards, which plays its part. A number of observers testify to its existence; and as the ventricles shorten during systole, general downward movement seems a matter of necessity, otherwise, instead of impulse against, there must have ensued recedence from, the parietes: the systolic swelling of the substance of the ventricles obviously would not suffice to counterbalance the coëtaneous diminution of their length. Elongation of the great vessels is probably concerned in this systolic lowering of the heart's position.

70. The normal impulse, then, seems to be a resultant of bulging of the ventricles, and of locomotion of the heart in two different directions, downwards and sidewards.[2] And the general laws of morbid action justify us in looking for varying degrees and combinations of these three elements of the impulse in disease.

B.—IN DISEASE.

71. I. *Impulse.*—(*a.*) *In non-cardiac disease.*—In adynamic diseases, and in various non-febrile blood-diseases, attended with depression of vital force, the heart's impulse habitually loses its strength.

Certain affections of the brain and spinal cord weaken the force of the heart's impulse; as do also various medicinal substances—aconite, digitalis, hydrocyanic acid.

Consolidation of the adjacent edges of the lungs, or abundant solid induration-matter in the contiguous pleura, solid substances in the mediastinum, and upward enlargement of the liver or spleen, lead to exaggeration of the heart's impulse; solid material being better fitted for conducting the heart's motion than spongy lung. Diminished size of the lungs, especially of the left, and pleuritic adhesions so placed as to withdraw the edges of the lungs from in front of the heart, or to cause by pressure atrophy of the inclosed pulmonary parenchyma, intensify the palpable shock for obvious reasons. The falling in of the chest-wall, which ensues on these conditions of the lung and pleura, acts in the same manner. In all these cases, it will be observed, the exaggeration in force, and extension of area are *surface-appearances*, and do not indicate any real increase of either on the part of the heart itself; many a supposed hypertrophy of the heart is nothing more than a simulation of the disease by some one of the physical conditions now referred to. On the other hand, emphysema and hypertrophy of the lungs, especially of the left, by bringing an unnatural amount of pulmonary tissue between the heart and parietes, weaken the palpable impulse, and may completely mask the direct parietal shock of well-grown cardiac hypertrophy.

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[1] Various observers might be referred to in support of this change of form of the ventricles; the observations of Dr. Mitchell (loc. cit.) in his case of ectopia cordis are, however, enough for our purpose.

[2] The weight of evidence seems to show that the alleged forward movement in systole is nothing more than anterior surface-pressure from swelling of the ventricles.

Again, the quantity and quality of circulating blood affect the heart's shock: at least the excited and sharply forcible impulse, occurring during reaction after hemorrhage, appears in some part (perhaps frequency and force of propulsion in lieu of quantity propelled) due to the diminution of blood at command. In the spanæmia of chlorosis, the impulse, though sharp rather than strong, is yet stronger than in health. The condition of the heart's substance does not explain this.

72. (*b.*) *In cardiac disease.*—The diseased states of the heart which produce *real increase* in the force and area of impulse are—morbid functional excitement (all the varieties of purely dynamic palpitation, angina pectoris, the paroxysm of "cardiac asthma," &c.), inflammation (cardiac and peri or endo-cardial), and enlargement, especially if combined with adhesions of the pericardium. The influence of inflammation is merely functional and dynamic; increased impulse only exists at the outset of pericarditis, before effusion has occurred to any extent. The influence of enlargement, on the other hand, is organic and statical: pure hypertrophy increases to its maximum the force of impulse; hypertrophy with dilatation, the force and area combined; dilatation weakens force, extends area.

The impulse may be increased to such a degree as to shake forcibly the head placed on the stethoscope, nay, even to shake the entire body of the patient, and the bed on which he lies; and it may stretch diagonally from the eighth left rib to the first right interspace near the sternum, and transversely, in the third and fourth interspaces, from two inches to the right of that bone to as many outside the left nipple: in such cases the impulse is very distinctly perceptible even in the left back. Between this, the maximum amount almost ever witnessed, and the natural state, all possible gradations are observable. Increased force, when extreme, is always combined with other modifications of impulse.

73. The force of impulse is *lessened* by effusions in the pericardium—more readily in cases of passive than inflammatory accumulation, because in the former no excitement of the heart exists. Dilatation weakens the shock; fatty infiltration with softening has the same effect, although, on account of coexistent enlargement, the positive amount of impulse be above the average of health; abundant deposition of fat under the cardiac pericardium generally enfeebles the impulse.

74. Increased impulse may be especially perceptible either behind the lower part of the sternum from the fourth to the seventh left cartilage, and at the epigastrium—or below and about the left nipple, and between this and the left costal cartilages; in the former case the right ventricle is commonly most affected, in the latter the left. But exceptions to this rule occasionally occur; extreme relative hypertrophy of the right ventricle will, in addition to the impulse on its own proper side, produce so much to the left of the sternum, as to simulate that of enlargement of the left half of the organ.[1]

75. If increased impulse be mainly traceable in a horizontal direction, the right ventricle is by some observers held to be the source of the increase, because enlargement on that side has a tendency to render the position of the heart more completely horizontal; whereas if mainly traceable in a vertical, or vertico-diagonal direction, the left ventricle is at fault, inasmuch as its hypertrophy elongates the organ in those directions. I have occasionally found the former, frequently the latter, of these propositions correct; but neither is worthy of implicit clinical trust. Hypertrophy of the left ventricle, when extensive, most certainly widens the impulse horizontally.

76. The character of the impulse varies as much as its force. The *apex-beat* may retain the natural impulsive and gliding character, and be merely increased in power. Or it may acquire a quick, abrupt sharpness; or convey the sensation of slow heaving, or pushing forwards against an obstacle: in hypertrophy of the left ventricle, the latter character is habitual; in dilatation the former. Where weakened, the loss of power may be simple; or the shock be also abrupt and jerking, or marked by fluttering unsteadiness, as in various species of softening. The *general impulse* on either side of the heart may vary similarly.

77. In rhythm, the heart's natural impulse, we have seen, is synchronous with the systole of the ventricles, and single. Now the impulse lags slightly behind the systolic sound of the heart, where fluid is accumulated in the pericardium, and the position of the apex is *not* elevated; before the impulse reaches the chest-wall, a

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[1] Kernis, ætat. 10, U. C. H., Females, vol. ii. p. 237, 1847.

stratum of fluid must be pushed aside. Besides in certain states of disease, the shock becomes double, the added impulse being systolic or diastolic. Double *systolic* impulse is an occasional, though rare, attendant on eccentric hypertrophy, a not uncommon one (it may even be treble, whence a peculiar jogging, shaking character) where, in addition to the hypertrophy, the pericardial surfaces are agglutinated, or covered with recent exudation-matter[1]—more especially if the præcordial pleural surfaces be also adherent. Laennec noticed that in cases of great hypertrophy it seems "as though the heart swelled, and applied itself to the parietes at first by a single point, then by its whole surface, and finally sank back in a sudden manner." The sudden "sinking back" of the heart, Hope was the first to state, sometimes terminates in a jog or shock (back-stroke) obviously *diastolic* in time. He believed this "diastolic impulse" was strongest in dilated, though sometimes very considerable in simple, hypertrophy—and imperceptible in simple dilatation: this accords with my experience also. The explanation of the phenomenon given by Hope, seems, however, scarcely intelligible. "It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back terminates in a jog or shock, *occasioned by the refilling of the ventricles*, and constituting the diastolic impulse in question." But the mere expansion of dilated hypertrophous ventricles is doubtless in itself capable of conveying a forcible impulse to the surface; the intense power of the natural diastolic expansion has always been noticed by vivisectors. On the other hand, there are many cases in which the diastolic sensation is rather one of *inward succussion* than *parietal impulsion;* it then depends, quite as probably, on falling back of the weighty heart against the spine—a view strengthened by the fact that "diastolic impulse" is peculiarly obvious in cases where solid accumulations, aneurismal or other, lie behind an enlarged heart.[2] A *double* diastolic impulse has been described; this I have not observed—it appears not improbable that impulses, so spoken of on the ground of their being unattended with arterial pulse, were in reality systolic, but too weak to reach the radial arteries.

78. In the normal state, any successive number of impulses are equidistant, and of precisely identical, or very nearly identical, force; in diseased conditions of the organ this uniformity may disappear. There may be several strong shocks succeeded by others as feeble; and the number of each kind may be uniformly, or very nearly so, the same—or their number may vary greatly—in point of time all forms of irregularity are observable: force is rarely

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[1] Courtenay, U. C. H., Females, vol. vii. p. 303, et seq.

[2] When a heart either quite healthy, or affected with enlargement so slight as to be practically insignificant, is excited by exercise or emotion, a slight jog synchronous with the diastole may often be felt. But this jog cannot, as it appears to me, be correctly spoken of, with Dr. Bellingham, as an impulse: the impulsive character seems to be wanting.

thus affected without time being implicated also. Simple hypertrophy of one or both ventricles does not produce this perversion of rhythm and force, unless some condition seriously affecting the pulmonary circulation occurs—especially congestions, and inflammations of the lung. But both force and time are affected to a high degree, when dilated or simple hypertrophy of the left ventricle coexists with mitral insufficiency; force more than time, where the same state of the ventricle attends aortic insufficiency. Marked dilatation, fatty infiltration, fatty metamorphosis, and mere flabby softness of the heart, produce great irregularities of rhythm and force of impulse—but in these cases no single impulse is, absolutely speaking, powerful, unless the walls of the ventricles be thickened also. Pericarditis, both before and after the occurrence of liquid effusion, is occasionally attended with similar irregularity; so, too, are various malformations of the heart, especially during paroxysms of dyspnœa; here perversion of the relative capacities of the two sides of the heart probably acts as the immediate cause of the irregularity.

79. The impulses, whether natural or morbid, hitherto spoken of, are caused by ventricular action: does the contraction of the auricles produce visible or palpable impulse? Certainly not, in the state of health: their systole is feeble, and tends to withdraw them from the surface; their diastole yet feebler, passive and gradual in character. Where, however, the auricles are hypertrophous and dilated, it is conceivable that their systole, perhaps even their diastole, may become perceptibly impulsive. Auricular impulse, if systolic, should very slightly precede the shock of the heart's apex; if diastolic, it might coincide with any moment of the ventricular systole, or diastole, and, in all probability, actually would coincide with the commencement of the former.

In a case where percussion-dulness at the second left interspace indicated an enlarged left auricle, impulse, preceding the ventricular systole, existed in the third left interspace close to the sternum; this was probably auricular and systolic. The left ventricle was hypertrophous, and the mitral valve insufficient.[1] It is probable, too, as suggested by Dr. Blakiston,[2] that the impulse of the ventricles may be communicated, in some cases of dilatation of the appendix of the left auricle, to the second left interspace: such *pseudo-auricular* impulse would synchronize with the ventricular systole.

80. II. *Form.*—The præcordial intercostal spaces are, in the natural state, of the same form and dimensions as the corresponding ones on the other side of the chest. They are widened by hypertrophy—widened, and even bulged outwards, by pericardial effusion, especially in the young; narrowed by chronic pleurisy and

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[1] Frederick Smith, U. C. H., Males, vol. v. p. 296. Oct. 1850.

[2] Diseases of the Chest, p. 124.

pericarditis, particularly if there be no hypertrophy sequential to the latter.

81. III. *Movement.*—The state of movement in these spaces is better appreciated by the eye [56] than by the hand.

82. IV. *Vocal Fremitus.*—Where the heart is uncovered by lung, vocal fremitus is more or less completely annulled; the edges of the lungs at the cardiac region may hence be distinguished by means of this sign with a good deal of precision—that of the right organ more satisfactorily than the left as a general rule.

83. Alteration in the area of deficient vocal vibration over the heart-region is a sign scarcely appealed to in the diagnosis of cardiac affections. Perhaps it deserves to be more frequently employed. It cannot be questioned, for instance, that enlargements of the organ, especially of its right division, and accumulations of fluid in the pericardium will annul the fremitus to an unnatural extent to the right of the sternum. But the other signs of these states are numerous and positive; and, as far as I know, the only conditions under which this defect of fremitus might prove clinically valuable, would be where dull percussion-sound of undetermined nature existed at the right border of the heart. For, if this toneless percussion depended on induration of the lung, the vocal fremitus would be increased instead of being annulled or weakened.

84. *Adventitious phenomena.*—Application of the hand detects, under special circumstances, two kinds of phenomena which are purely adventitious—valvular thrill and pericardial friction-fremitus.

85. (1.) *Valvular thrill*, or "purring tremor" resembles, in many cases, the vibration of the surface of a purring cat—in others is more like the vocal fremitus felt over the larynx of persons with powerful bass voices. Varying in force in different cases and in different conditions of disease, it may change from moment to moment in the same person, according to the energy of the circulation—mental or bodily excitement rendering it powerful, when, in the state of repose, it had been almost imperceptible. Synchronous, and agreeing in rhythm with the ventricular systole, it is felt mainly below and within the nipple, about the fourth interspace over an area of from one to three inches; or at mid-sternum on the level of the third rib, in the third left, and the second right interspaces close to that bone: if perceptible higher than these limits, and especially if so above the clavicle, this species of thrill ceases to be of purely cardiac origin. Habitually caused by forcible and rapid propulsion, in a rippling current, of blood—more particularly of blood altered in certain of its properties—through orifices narrowed and roughened by disease, it may in rare cases occur independently of textural change in the valves. When connected with valvular disease, it ceases, for obvious reasons, to be produced, unless a certain quantity of blood is propelled with a certain force through the diseased orifice; and hence, after existing to a high

degree, it may disappear, because the narrowing which originally led to it has increased to an extreme amount, while the heart's force has, from some independent cause, more or less failed; a similar alteration, it is well known, occurs previously to death in certain organic murmurs of the heart.

The two combinations of disease, in which cardiac thrill is observed to the maximum degree, are insufficiency of the mitral valve with dilated hypertrophy of the left ventricle—and constriction of the aortic orifice, coupled with hypertrophy of the same kind and seat: in the former case the seat of the phenomena is below and within the nipple; in the latter, at the aortic base. Now, the physical conditions of the phenomenon seem pretty fairly balanced in point of completeness in these two combinations; yet certainly cardiac thrill is more frequently met with as a dependence on mitral regurgitant than aortic constrictive disease. Hope supposed that this arose from aortic thrill being masked by the interposed sternum; but, although it is true, thrill, thus localized, may be sometimes rendered more distinct by causing the patient to lie on the left side, I believe the real cause of the comparative rarity to be no other than the less frequency of marked aortic than of mitral disease.

I do not remember ever to have observed cardiac thrill synchronous with the ventricular diastole; tricuspid and mitral constriction,[1] as well as aortic regurgitation, have consequently not in my experience led to its production. The force of the current is, perhaps, insufficient in the three cases: yet I can well conceive that in a spanæmic person, with highly developed aortic regurgitation, a minor degree of the phenomenon might exist; a loose vibratile portion of valve or vegetation, floating in the current, might also greatly facilitate its production. Neither have I ever found acute endocarditis, where there was full certainty of the absence of old-standing valvular disease, attended with thrill. But I have occasionally known a faint systolic aortic thrill, never a mitral one, accompany palpitation in spanæmic women, and in persons who had suddenly lost a large quantity of blood. I do not believe that mere nervous excitement of the heart will produce thrill in people whose blood is healthy.

87. (2.) *Pericardial tactile friction-fremitus*, like the analogous phenomenon of pleural fremitus, is considerably rarer, and, when occurring, of shorter duration than audible friction-sound. It has much more of a rubbing character than valvular thrill, with which it may coëxist, and is more movable than this, migrating from one part to another of the cardiac region within the course of a few hours, although the patient has retained and is examined in the same posture.

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[1] If mitral constriction be present, the thrill of coexistent regurgitation will be thereby rendered more intense.

In doubtful cases pericardial may be distinguished from ordinary pleural fremitus by causing the patient to suspend his breath, for its rhythm is of course cardiac;[1] an excess of fremitus commonly attends the ventricular systole. Cardiac action may be supposed capable of producing pleural tactile fremitus, as it indubitably does audible intra-pleural friction sound [239]; but I do not remember ever to have observed the fact: the diagnosis would be very difficult.

A remarkable case has been recorded by Dr. Swett,[2] where a distinct "thrill" over the heart was caused by friction against an enlarged and "tuberculated" left lobe of the liver; the heart, as likewise the pericardium, proved "perfectly normal in all its parts." Cardiac action may then produce tactile fremitus beyond the pericardium, and even through the diaphragm.

88. But setting aside these exceptional modes of mechanism, and admitting that a given vibration, felt over the heart, is really produced either within the organ, or on its pericardial surface, is it possible to localize the phenomenon with confidence in one or other of these sites? In the majority of cases the diagnosis may be securely made by the intrinsic quality of the sensation itself—in the one state a series of rubs, in the other a single thrill. This mode of determination may fail; numerous associated characters, very much like those available in the distinction of peri- and endocardial murmurs, will then afford help through the difficulty: valvular thrill will be distinguished from pericardial fremitus by its apparent depth from the surface, its steadiness of force, its uniformity of position, its maximum force in the same site as that of valvular murmurs, its non-increase by bending the trunk forwards, its precise synchronism with the heart's systole, perhaps in rare instances with its diastole, and lastly the coëxistence of valvular murmur. Characters more or less accurately the reverse attend pericardial fremitus.

SECTION III.—MENSURATION.

89. Mensuration of the surface corresponding to the heart, confirms inferences, otherwise deduced, as to the existence of certain affections of the organ; and sometimes becomes a useful auxiliary in diagnosis.

90. Thus, in health, the nipples are equidistant from the middle line; in enlargement of the heart and in pericardial effusion, the distance between the left nipple and that line undergoes increase—to a very notable amount in some old-standing cases of dilated hypertrophy. The left nipple, too, is carried somewhat downwards by the same affections. Again, the space comprised between the

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[1] Such difficulty must, however, be rare: pleuritic fremitus produces the sensation of slow and deliberate, pericardial, that of quick and abrupt, rubs.

[2] New York Journ. of Med., July, 1840, p. 6.

upper border of the third, and lower border of the sixth ribs, an inch outside the sternum, is the same on both sides in healthy persons free from curvature of the spine: the diseases just named increase the measurement on the left side. The influence of the two affections is the same on these measures; it differs in regard of the following one. The vertical distance between the left clavicle and the heart's apex-beat is in health the same as between the right clavicle, and the point on the right side, lying on the same horizontal level as, and corresponding to, that beat; now, in pericardial effusion, the distance on the left side falls considerably below, in enlargement of the heart rises considerably above, that on the right side. This admeasurement is really useful in some cases of difficulty—as, for instance, when an enlarged and dilated feeble heart beats with a quasi-undulatory impulse.

91. In forced inspiration, the expansion on the level of the sixth cartilage is in health slightly less on the left side than the right, a deficiency referable to the influence of the heart. In pericardial effusion this deficiency, instead of, as might on first thought be supposed, increasing, actually disappears; the pressure of the fluid on the diaphragm throws an extra amount of work on the ribs, which comparatively move more than in health. When the fluid accumulates to a great amount, however, say twenty ounces, the præcordial ribs, also, move less than in health. I do not know how this matter stands previous to the occurrence of effusion in pericarditis; nor have I sufficiently examined the point to say positively how the costal motion varies in the different varieties of enlargement of the heart, with and without valvular disease.

SECTION IV.—PERCUSSION.

A.—IN HEALTH.

92. Numerous difficulties stand in the way of accurate discrimination of the heart's outline by percussion. Above the organ lie bloodvessels of large calibre with their contents, forming a quasi-solid mass; and, besides this, the variable amount of fat and areolar tissue occupying the mediastinum may prove a source of serious clinical difficulty. Again, the liver, furnishing a percussion-note but slightly differing in amount, resonance, and pitch of tone from its own, coasts the right inferior border of the organ. Furthermore in front of the heart are placed the sternum, costal cartilages and ribs, possessed of a special resonance. Add to all this, that the organ is in a state of perpetual restlessness, and constantly changing its precise axis, bulk, form, and mode of relationship to the surface, while the quantity of lung in front of it on either side is likewise undergoing the changes dependent on respiration. Yet, in despite of all these obstacles, it is possible by care to establish, with sufficient accuracy for all practical purposes, the position and bulk of the organ;—nay, to measure approximatively its thickness.

93. The heart cannot be percussed with success except in the recumbent posture; and, unless for the special purpose of ascertaining to what extent the organ (or the source of præcordial dulness, whatever it be) is movable, not the slightest movement, even of the limbs, ought to be permitted during the examination. The force used in striking will vary from the gentlest touch with the flat surface of the pulp of the middle finger, to a sharp tap with the ends of one or more fingers; it is a mistake to suppose that violent blows serve any useful purpose.

94. Percussion of the chest, where the heart lies immediately beneath the surface, gives a short, dull, high-pitched sound; the parietal resistance is highly marked. These characters are modified very perceptibly, except in the intercostal spaces, by the special resonance of the sternum, ribs, and cartilages. The sound emitted by these textures is notably more prolonged, of greater resonance than, and of different pitch from, the cardiac; and when the heart happens to reach only just to the right edge of the sternum (so that the proper tonelessness of wet muscular substance and intra-cardiac blood cannot affect the percussion-results to the right of that bone in the right half of the chest), it may prove next to, if not absolutely, impossible to define with confidence the outline of the right ventricle. I believe that the texture of the sternum is in some persons peculiarly resonant; there are, doubtless, too, interfering acoustic conditions, one of the most indubitable of which, *horizontal conduction*, will by-and-by be dwelt upon.

95. Where the organ is invested in front by lung, the resonance partakes of course, more or less, of pulmonary quality, according to the thickness of the intervening stratum of pulmonary tissue. We may then clinically speak of the heart's *superficial* and *deep-seated* dulness.

96. By reference to the Diagram (page 18), it will be seen that the limits of the heart's *superficial dulness* must be as follows: on the right a vertical line, extending at midsternum from the level of the fourth rib to that of the sixth; on the left an oblique line passing outwards and downwards at a more or less acute angle from the latter, opposite the fourth cartilage, and curving inwards again, somewhat within the site of the nipple,.to the sixth rib, beside the heart's apex; inferiorly, a line gently sloping to the left, from the central point of the lower edge of the sternum, along the sixth cartilage. This is the extent of heart uncovered by lung in calm respiration; and the form of the part is obviously, though only rudely, triangular: the lengths of its sides have already been stated [12]. The area of the triangle is to be made out only by the gentlest taps with the pulp of a finger, either on the surface directly, or on a finger of the other hand, used as a pleximeter. The right border is with difficulty established, on account of the sternal resonance.

The outlines of this islet of superficial dulness vary with the conditions of the act of respiration. Forced inspiration diminishes the extent of its area; expiration increases that extent, especially in an upward direction and to the right; and by the kind of percussion described above, these changes in dimensions can without much difficulty be ascertained. They are, however, more frequently serviceable as tests of the freedom of play of the edges of the lungs, than of the condition of the heart.

The triangular shape of the dull-sounding space, it must be mentioned, not only undergoes change with the different periods of respiration, but is sometimes inclined to squareness in outline, and sometimes shapeless.

97. Beyond the limits of superficial dulness, the outline of the heart may be ascertained by the deep-seated dulness elicited by firm percussion. This *deep-seated dulness* extends normally in a vertical direction, from the third to the edge of the sixth cartilage, and transversely from the left nipple to a little beyond the right edge of the sternum opposite the fourth cartilage; the longest measurement is the diagonal one, from the upper part of the third right cartilage (the right auricle) to the point of the apex-beat. In ascertaining these limits there are two main sources of perplexity. The first consists in the difficulty of defining exactly the line of the base; as the dulness, produced by the mass of large vessels there, is nearly as great as that of the heart itself: this perplexity, fortunately, only occurs where the edges of the lungs are unusually far apart. The second lies in the difficulty of separating the adjacent edges of the heart and liver. The pitch of the liver-sound is higher than that of the heart *in situ* (especially if *horizontal conduction* from tympanitic intestine does not modify the proper hepatic resonance); and in many cases ordinary percussion will succeed in eliciting a sensible fall in pitch as we pass diagonally from the liver to the heart. Sometimes a narrow space, say from a third to half an inch in width, may be detected, giving a slightly tubular note, and this space, easiest detected if the patient be made to keep his mouth open, while percussed, corresponds to the line of union of the two organs.[1]

The "auscultatory percussion" of Drs. Cammann and Clark[2] most certainly renders the change of pitch, on passing from the liver to the heart, more positive and definite than it appears under ordinary percussion, and may be called to our aid in puzzling cases. The method referred to consists in receiving percussion-sounds directly through a solid stethoscope to which the ear is applied, instead of, as in the ordinary way, receiving them weakened by diffusion through the air. By practice the process becomes manageable without the assistance of a second person; the observer, having placed his ear to the stethoscope over the lower part of the liver, percusses the surface diagonally upwards and to the left, in the direction of the heart, and is apprised of his reaching that organ, by abrupt change in the pitch of the sound. The interesting paper of the American physicians is well worthy of study.

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[1] I presume this is because at this line interruption of media takes place.

[2] New York Journ. of Med., July, 1840.

98. Certain physiological conditions modify the exact area of the heart's dulness. Its outline cannot be identical during the different periods of the heart's revolution: the chances that percussion gives the outline in the state of ventricular systole, diastole, or of quiescence, may be represented respectively by the numbers 2, 1 and 2. The chief interest of this matter turns on the testing the correctness of results, obtained during life, by the actual size of the organ after death; it may be neglected in ordinary practice.[1] Again, the act of respiration, and alterations of posture of the trunk, will of course change the position of the heart's dulness. The area of superficial dulness is relatively less in infancy, greater in old age, unless the lungs become emphysematous, than in manhood.

99. And, further, the acoustic phenomenon which I have designated as *horizontal conduction*[2] may exercise a very notable and deceptive influence on the apparent area of the heart as defined by percussion. When moderately strong percussion is made, even on the central point of the heart-region [11], the impulse horizontally conveyed to the stomach, especially if that organ be pretty fully inflated, may elicit a response in the form of a somewhat tubular note mingling with the proper dull, toneless resonance of the cardiac substance.[3] This horizontal conduction is not to be confounded with the well-known vertical conduction of the sound of a deeper-seated organ through another lying over it—as when hepatic dulness mingles with pulmonary resonance in answer to percussion in the fifth right interspace. In the heart-cases referred to, we must first assure ourselves that the stomach does not lie behind, be it ever so deeply, the spot percussed; or if this be impossible, we must percuss so gently as to make it impossible the blow could influence a deeply-seated organ.[4]

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[1] The heart is drawn up after death, both by the emptying of its cavities and by the expiratory collapse of the lungs: hence another of several reasons why slight differences may arise in clinical and post-mortem examinations. The mode of death must seriously influence this.

[2] Diseases of the Lungs, 3d Am. ed., p. 78.

[3] Aubrey, U. C. H., Males, vol. xvii. p. 268.

[4] This horizontal conduction has an important bearing on the percussion-results of other organs. M. Aran, in his translation of Prof. Skoda (p. 409), criticizes his author sharply for affirming that the "percussion-sound in the renal region may be completely dull when the kidneys are very small, and tympanitic when they are very large." Skoda proffers no explanation on the matter. Now I have had occasion to notice a case fully demonstrating the truth of the latter clause of Skoda's statement; to the other M. Aran, of course, would not object. In a man with an enormous cancerous mass occupying the entire right half, and encroaching somewhat on the left half, of the abdomen, the right renal region gave amphoric resonance under percussion; and this circumstance, taken in conjunction with the facts that no intestine lay in front of the tumor, and that the urine was of normal

B.—IN DISEASE.

100. I. *Non-Cardiac Disease.*—Various morbid conditions, independent of disease of the heart or its membranes, may *increase* the area of præcordial dulness. Among these may be enumerated pure atrophy of either lung, with diminished volume; consolidations of the portions of lung adjoining the heart; enlargement and elevation of either lobe of the liver; accumulations in the mediastinum or pleura; diminution of bulk of the left lung with lateral pleural adhesions, a state tending to bring an undue quantity of the heart into contact with the chest-wall; aneurism of the great vessels; and even, in infinitely rare cases, tumor, with constriction and pouching, of the œsophagus. In all these cases, the actual physical state of the heart may be completely unaffected. Not so, when the extent of præcordial dulness is increased in consequence of the stagnation of blood in the right cardiac cavities, caused by obstruction of the pulmonary circulation in various forms of dyspnœa; here the heart's own dimensions are temporarily changed.

101. There is only one thoracic affection independent of the heart itself which seriously *diminishes* the extent of præcordial dulness—emphysema of the lungs—particularly of the left, and especially when conjoined with bronchitis. The temporary influence of bronchitis in increasing the bulk of an emphysematous lung, and so masking the dulness of a very greatly enlarged heart, is well seen in cases, where, superadded bronchitis yielding to treatment, the area of cardiac dulness undergoes sensible increase from the removal of pulmonary distension.[1] Conversely it may be seen that ascites diminishes the extent of the heart's dulness, by pushing the organ upwards under cover of the lung.

Venesection, as first shown by M. Piorry, will very sensibly diminish the extent of præcordial dulness, especially towards the right side, in persons whose right cavities had previously been loaded with blood. A marked state of anæmia, by reducing the heart's distension, narrows the area of its dulness.

102. II. *Cardiac Disease.*—The area of præcordial dulness may be *diminished* or *increased* by disease of the heart itself.

103. Diminution of that area attends primary concentric atrophy of the organ; but though decrease in bulk of the heart, and in calibre of the large vessels, often occurs to a very notable amount

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characters, seemed to connect the mass with the liver. Yet the tumor proved to be wholly renal—the intestines were pushed in a body far away into the left abdomen, and the right ureter being obliterated, healthy urine flowed from the healthy left kidney. And, further, the substance of the morbid mass lay in close contact with the abdominal lumbar wall in the very site of the amphoric resonance. Now, as no intestine lay between the tumor and the anterior abdominal wall, the special amphoric intestinal tone must have been horizontally conducted through and along the vertebral column from the coils of bowel far away in the left half of the abdomen.—Bowles, U. C. H., vol. xvii. p. 114.

[1] Case of Hope, Clin. Lect., "Lancet," loc. cit. p. 443, 1849.

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in the tuberculous and cancerous cachexiæ, it is seldom detected during life in either: adjacent pulmonary dulness renders its discovery difficult in the former case; it is seldom sought for in the latter.

In pneumo-pericardium the natural dulness disappears more or less completely in proportion to the quantity of gas accumulated; even if there be fluid, as well as gas, in the serous sac, the entire præcordial region may give more or less tympanitic resonance in dorsal recumbency. By changing the patient's posture from the back to the side, dull sound will be elicited in the then inferior, tympanitic sound in the then superior, part of the præcordial region: this interesting fact I succeeded in establishing in a singular case of traumatic communication between the œsophagus and pericardium.[1]

104. The cardiac affections which *widen* the area of præcordial dulness are materially more important than those just reviewed; they are referable to three main heads: enlargement of the heart, and fluid accumulation or solid formations within or about the organ.

105. Hypertrophy, in all its observed forms and sites, increases the extent of cardiac dulness: the position of the increase and the elasticity of the walls differ under different circumstances. Simple hypertrophy of all the cavities, or of both ventricles, or the same state combined with uniform dilatation, extends the outline of dulness downwards, and to both sides, more, however, to the left than the right: scarcely any impression is produced on the chest-resonance at the upper outline of the heart—a proposition which applies, with scarcely an exception, to all varieties of true cardiac enlargement; for, as already mentioned, the tendency of increase of bulk is to carry the heart downwards. In general dilatation, simple, or combined with attenuation, extension of dulness is also observed; and very careful percussion will substantiate the existence of less parietal resistance in this than in the preceding cases. If enlargement be limited to either ventricle, the extension of the dulness takes place in the direction of the affected one; and what has been said concerning the site of impulse in such cases [74], applies to that of dulness. Accumulation of fat under the pericardium, when sufficiently great to alter the limits of dulness, generally does so to the right side; for the simple reason that such accumulation begins with, and attains its maximum at, that side. The influence of fat in this manner is, however, very rarely to be observed. Hypertrophy of the left auricle carries deficiency of resonance into the second left interspace.

If with the enlargement coëxist close pericardial adhesions, the extent of dulness is always proportionately increased, but more so to the left than the right side: this effect is still more perceptible if there be pleuritic agglutination in the left infra-mammary region. In some cases of old pericarditis the area of the heart's dulness is increased upwards: thus I have repeatedly found, in persons who had previously been under my care for rheumatic pericarditis, that more or less marked dulness existed, years after, in the second left interspace, even up to the second cartilage, and at the adjacent part of the sternum. This state of resonance may exist with or without obvious enlargement of the heart; if without such enlargement, it can only be explained by the presence of solid induration-matter about the great vessels and base of the heart; if with such enlargement, it is explicable by the elevation, which the heart undergoes during the effusion-period of pericarditis, being maintained by agglutination of the pericardium, in spite of the depressing influence of the enlargement.

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[1] Case of Ramo Samee, U. C. H., under the late Dr. A. T. Thompson. The perforation was produced in the attempt to swallow a long blunt instrument, a juggler's "knife;" the case terminated fatally, and the preparation is in U. C. Museum—No. 3859.

106. Fluid accumulation in the heart's right cavities (of blood, of course), occurs to sufficient amount, under certain circumstances of obstructed cardiac circulation, to extend very perceptibly the area of dulness on the right of the sternum. The extension of dulness takes place mainly between the second and the fifth interspaces. The most important condition of the kind, practically, is dilatation of the right auricle and ventricle, combined with insufficiency of the tricuspid valve. But the accumulation of fluid and semi-solid blood in endocarditis sometimes considerably widens the area, both to the right and left; and where obstruction by fibrinous coagula is gradually effected in the pulmonary artery, a like gradual increase in the area of dulness to the right of the sternum, immediately dependent on consecutive clotting of blood in the right cavities, may clinically be established.[1]

107. Fluid accumulation in the pericardium, even when of small amount, and whether passively or actively dropsical, or the result of pericarditis, produces an extension of cardiac dulness. I know that four ounces of liquid will widen the area of dulness—perhaps even less than this will suffice. Clinical experience proves (the results of artificial distension of the pericardium obviously could not be accepted as conclusive evidence) that the pericardium undergoes distension most readily upwards, with greatest difficulty downwards, with medium facility forwards, backwards, and sidewards. When distended with fluid, the sac retains its original rudely pyramidal form—the base below, the apex above. The level of that *base* at the front of the chest commonly corresponds to the lower border of the sixth rib, sometimes to the sixth interspace, in very rare instances to the seventh rib: in the latter class of cases the texture of the pericardium had probably undergone some chronic change, of a rarifying kind, before the occurrence of effusion—or

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[1] Case of J. S., seen with Dr. Markham.

at least been distensible with more than average facility by fluid pressure. In cases of extreme accumulation the diaphragm is arched downwards by the fluid; the epigastrium may thus be rendered somewhat prominent, and the dulness of the fluid is with difficulty distinguishable from that of swollen liver—it does not reach sufficiently to the left side to modify the percussion in the hypochondrium. The *apex* of the pyramid, as the fluid increases, gradually rises to the second left cartilage, to the first, to the sterno-clavicular joint, and even to nearly an inch above the clavicle, displacing the apex of the lung at the inner aspect of the supra-clavicular region. As the fluid increases, it pushes aside the edges of the lungs, where they join in inspiration at mid-sternum, between the second and fourth ribs;[1] and this sideward detrusion, in cases of abundant effusion, condenses the adjacent edges of the lungs, and so increases the lateral extent of dulness. On the right side of the distended sac the state of vocal fremitus will sometimes guide to the line, where the condensed tissue and the fluid join—strong over the former, feeble or annulled over the latter.

With equal superficial extent the dulness from pericardial fluid is more absolute, and the parietal resistance greater, than from hypertrophy; a fact probably depending on the more perfect approximation of the fluid, than of the solid, material to the walls. This distinction is too delicate to be trusted to at the bedside; the dulness of effusion is better distinguished from that of general hypertrophy by its extensive range above the third rib, and its ordinary limitation to the sixth rib below; and from dilated hypertrophy of the right or left ventricle by not extending, disproportionately to the area of dulness, towards the right or left side. It must not be forgotten that an aneurismal sac (we may suppose it filled with fibrin, pulseless, latent, and *pro tanto* deceptive), of the transverse part of the arch of the aorta, and bulging inferiorly; or a small mediastinal tumor; or even superabundance of natural fat, placed just above the third left cartilage, and behind the sternum (a source of fallacy more frequent in persons with much subcutaneous fat than in the thin); may all three give to the dulness of a simply enlarged heart the triangular outline of that dependent on effusion in the serous sac. In all the instances referred to the dulness of the heart itself possesses its own form, more or less closely that of a square or a parallelogram—the relatively narrow and pointed addition above, caused by one or other of the three conditions mentioned, leading to the simulation of the triangular outline of fluid collection. And if an enlarged heart, with such an accidental appendage above it, be weak and flabby, and give a quasi-undulatory impulse, the distinction of the case from one of pericardial effusion, especially if there be acute pyrexial symptoms and the commemorative history be imperfect, becomes one of most serious difficulty.

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[1] Bartlett, U. C. H., Males, vol. iv. p. 292. The lungs were *five* inches apart on the level of the second rib. Such extraordinary distension could only result from very slow progress of the disease: it appeared to have lasted in this case six weeks.

108. The plastic exudation-matter of pericarditis sometimes forms a layer, one-third to three-fourths of an inch thick, of solid substance applied to the heart's surface: if chance place this great thickness of substance on the lateral confines of the organ, the area of dulness must be proportionately increased; but the sign is one of too great delicacy to be clinically serviceable.

109. Cancerous accumulation in, or underneath, the pericardium affects the præcordial resonance in proportion to its amount.

110. The *quality* of the percussion-sound undergoes no very material change under the circumstances hitherto referred to, except in cases of pneumo-pericardium. In this affection the tone becomes tubular, amphoric, or genuinely tympanitic. Further, in those very rare morbid states, calcified induration of the heart's substance and pseudo-ossification of the pericardium (of neither of which have I had much clinical experience), the resonance would, theoretically, become more ringing and osteal in quality than natural, and at the same time the resistance of the parietes prove sharp and highly marked.

111. The *form* of *deep-seated* dulness, peculiarly affected, as we have seen, by pericardial effusion, remains essentially unchanged by general hypertrophy; excessive hypertrophy of any particular part must of necessity modify the character of the general outline, but rarely to an amount that can be defined at the bedside. Dilatation renders the shape more or less markedly square. Again, hypertrophy alters the form of the *superficial* dulness by pushing aside the lungs, and converts the small triangular, into a relatively large and irregularly square, space. Solid masses under the pericardium change the outline of dulness according to the direction of their growth; but their influence is very slight in this way, and on their rarity it is needless to insist.

112. Valvular diseases exercise no direct influence on præcordial resonance; the increase of dulness so frequently coexisting with them of course really depends on some form of attendant enlargement of the heart. The membranous inflammations, when acute, though probably slightly increasing the heart's bulk by congesting its substance, do not *per se* (endocarditis at any period, pericarditis until exudation has occurred), appreciably alter præcordial dulness.

113. Præcordial dulness, whether dependent on fluid in the pericardium or enlargement of the heart, moves from side to side somewhat, as the posture of the patient is changed. Old pericardial adhesions, even, will not prevent sideward locomotion of a hypertrophous heart—nor will pleuritic adhesions, in front of the heart in addition, do so, unless they be at once closely agglutinative and extensive.

SECTION V.—AUSCULTATION.

114. Auscultation, directed to the heart, analyses:—(I.) Certain sounds produced by the normal action of the organ; (II.) Modifications of these occurring independently of heart-disease; (III.) Morbid states dependent on heart-disease. Besides (IV.) the state of the respiratory murmurs, and (V.) of vocal resonance, specially in the præcordial region, sometimes affords useful information.

115. While the heart is auscultеd, the patient should, as a rule, be placed in the recumbent posture, with the head slightly raised; unless, from the nature of his disease, this posture be an uncomfortable one. As much of the precision of the notions, obtained from the examination, depends on our being able to connect the spots, where various sounds are heard, with certain parts of the heart itself or great vessels, it is obvious that, in order to both simplify our task, and give all attainable surety to the indications, we should habitually auscult patients in the posture in which clinical practice must present them most frequently to us.

116. If there be any doubt about the superiority of mediate or immediate auscultation, in the case of the lungs, there is none in that of the heart. Certain phenomena, well audible at a given point with the stethoscope, may cease to be perceptible *one-third of an inch* beyond that point: such limitation as this evidently could not be effected by direct application of the ear to the surface.

117. The heart's sound may be more or less masked by certain morbid states of the respiratory sounds—and they may be given unnatural characters by the chance coincidence of the sound of natural inspiration or expiration; hence the necessity of causing the patient to hold his breath from time to time, while the heart is auscultеd.

(I.) NORMAL CARDIAC SOUNDS.

118. Each complete revolution of the heart is accompanied by two successive sounds, audible by means of mediate or immediate auscultation in the præcordial region, and separated from each other by intervals of silence. These two sounds differ in all their characters—intensity, duration, pitch and quality: and the two periods of silence differ in the only character they can differ in, namely, duration. These differences are found, provided the posture of the patient and the position of the stethoscope be unchanged, to be maintained either without the smallest, or with very slight, variation in any series of beats of the same heart.

119. The first of these two sounds, coincident with the systole of the ventricles, the heart's shock against the side, and the pulse, or diastole, of the arteries nearly adjoining the heart, is called the
or systolic, or (because normally of maximum force at the
art of the cardiac region) inferior, sound of the heart. The
of the two sounds, synchronous with the diastole of the

ventricles, the recedence of the heart from the side, and the pulseless state, or systole, of the large arteries, is known as the second, diastolic, or (because of maximum loudness at the upper part of the cardiac region) superior sound of the heart. The noiseless period succeeding to the first sound may be called the first, or post-systolic silence; that succeeding the second sound, the second, or post-diastolic silence.

120. If the period of an entire revolution of the heart, that is from the commencement of one first sound to the commencement of the next succeeding first sound be divided into ten equal parts, the durations of the several periods of sound and silence will be found on an average very closely as follows:—

First Sound	$\frac{4}{10}$
First Silence	$\frac{1}{10}$
Second Sound	$\frac{2}{10}$
Second Silence	$\frac{3}{10}$

The total duration of sound would hence stand in the rates as 3 : 2 to that of silence. I base this estimate on the results obtained by careful auscultation of a series of healthy males; it is only to be taken as approximatively true, both because there are very sensible differences in individuals and because error is easily committed in estimating the extremely minute divisions of time concerned. Thus when the pulse beats as much as from eighty to ninety times in the minute, the post-systolic silence is difficult enough of detection; but it becomes obvious, where the pulse does not exceed sixty in a minute. The estimate, also, refers only, the student will remember, to the periods of sound and silence, not to those of action and inaction, of the heart: clinically there is no trustworthy plan of measuring the length of the systole and diastole.

Any very notable deviation from the healthy standard is easily enough appreciated by the ear; as, for instance, in certain cases of dilatation, where shortening of the first sound and lengthening of the first silence rarely fail to arrest attention.

121. An accurate idea of the characters of the heart's sounds cannot be given by a single description of them, as heard in any one particular spot: they vary materially at different parts of the cardiac region, both in their positive and relative properties. The least study of the healthy chest will convince the student that the description which it is the habit to apply to the sounds of the heart generally, holds good only when these are heard towards the left apex. The sounds require comparative analysis: (*a*) at both sides of the apex-region, and at both sides of the base-region; (*b*) at base and apex on the same sides of the organ; (*c*) at base and apex on opposite sides. Now this is the most difficult of all studies connected with the healthy heart; but it is essential as the basis of observation of the organ in a state of disease.

122. (*a*.) At the left apex the first sound is dull, measured, booming, prolonged, and strongly accentuated; its commencement pretty

sharply defined, its close much less so. Double the length of the second sound, of lower pitch than this, and seeming to the ear deep-seated, it attains its maximum at this apex, in regard of accentuation, prolongation, and measured, booming character, but not habitually in amount of sonorousness. The second sound, only half as long as the first—clear, abrupt, flapping, and short—is more sonorous, more superficial, and of higher pitch than its predecessor. At the right apex-region, the first sound is considerably clearer, shorter, more abrupt, less strongly accentuated, and of somewhat higher pitch than at the left apex-region. This difference of character depends, probably, both on the thinness of the walls of the right, as compared with the left ventricle, and on the parietes being of different conducting power in the two situations: at all events the sound, audible at the end of the sternum, may be fairly referred more particularly to the right ventricle; that near the nipple, to the left ventricle. So, too, the second sound is habitually clearer, and sometimes even of higher pitch, at the end of the sternum, than towards the left apex; this is, however, less constantly true, and I have known the converse to be the case, where no suspicion existed of the existence of dilatation of the left ventricle—where, indeed, the first sound possessed to the full its natural share of dull, prolonged, booming character.

Passing from the apex to the base, the same kind of dissimilitude, laterally, in the sounds, may be detected—not indeed precisely at the base, but a little above this, opposite the second interspace, where the aorta and pulmonary artery are in contact. The first sound at the right second interspace is commonly duller, of slightly lower pitch, and more prolonged than at the left corresponding point: in neither is it accentuated. The second sounds differ here in the same characters and in the same manner, but to a less degree: they are accentuated in both places, more on the right side than the left.

123. (*b.*) Let us now examine the two sounds comparatively at base and apex on the same sides of the heart. The first sound, strongly accentuated at the left apex—prolonged, booming, and dull—at the left base loses the accent, which passes to the second sound, while this becomes louder, more ringing, and sometimes even of higher pitch than at that apex. At apex and base on the right side, the characters of the first sound are very similar; it has more accent in the former than in the latter spot, however, both positively, and *à fortiori* as compared with the second sound.

124. (*c.*) The relative characters of the two sounds at base and apex at opposite sides of the heart (crucially taken, as it were) may easily be deduced from the foregoing account.

125. Now, if these statements be correct, it appears obvious that no single articulate symbol can be devised, applicable to the heart's sounds in more than one point. The subjoined series is offered for the four points of prominent clinical interest. The acute accent-

mark is used to show where the accent falls—twice, when it falls very strongly; the marks of long and short, where length or shortness of sound is a prominent characteristic.

	First sound.	Second sound.
At the left apex	ō̋ubb	dŭp
At the right apex	úp	tŭp
At the left base[1]	up	túp
At the right base[1]	ub	tŭ̋pp

The eye gathers from these symbols the tendency to equalization in length observable in the sounds at the bases, as well as the transference of the accent from the first to the second sound at the apices and bases respectively.

126. The extent, to which the heart's sounds are audible in health, is not subject to any fixed rule.[2] One great mistake, commonly committed by authors who attempt to define it, consists in their omitting to consider *separately* the first and second sounds. From this omission, the ordinary starting proposition, that "the heart's sounds are heard at their maximum in the præcordial region," becomes an error: the second sound is, in truth, heard in nine people out of ten more clearly at midsternum, on the level of the second interspace, than at any point of the præcordial region—even limiting that region to the space in which the heart is uncovered by the lung during tranquil breathing. The thickness of the soft parts, the form of the chest, and many other physical conditions perfectly independent of disease of any of the thoracic organs, modify the extent of propagation so variously, that there can be no practical utility in laying down rules subject to perpetual exceptions. But the lines of propagation of the two sounds severally agree in most healthy persons, whatever be their absolute intensity at their seat of production; changes in these lines point positively to some modifying cause, and hence their establishment is clinically valuable. Now, the first sound passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way; it loses much more on the route to, and at, the right acromial angle: its propagation backwards is clearest and fullest to the left—so that while audible at the left back, it may be inaudible at the right. The second sound, with the base-region as its centre, radiates to the right and left acromial angles, with greater clearness to the left than the right; posteriorly it reaches the surface at the right side less clearly than at the left.

127. *Mechanism of the heart's sounds.*—The difficulty of unravelling the mechanism of the normal sounds of the heart is broadly and emphatically proved by the fact that, from the time of Laennec

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[1] The left and right second interspaces close to the edge of the sternum.

[2] Some authors speak of the sounds being audible in healthy male adults, of moderate stoutness, even *at the right side posteriorly;* others write that the space over which they are heard *seldom exceeds two square inches in the cardiac region!* The truth is that both of these states exist in different individuals in perfect health: the error lies in regarding either of them as constant.

to the present day, some five and thirty theories, more or less completely differing from each other, have been proposed in its explanation. The subject seems at once a tempting and an exhaustless one. The zeal of experimentalists, unsatisfied with any one of the long series of conjectures previously proposed, seeks constantly to add something novel, either in the way of fundamental principle, or of combination of agencies already acknowledged, to the mass of acquired hypotheses. This zeal is commendable—and would be above all praise, were it not too often associated on the part of fresh investigators with a disposition to sneer at the attempts of predecessors as capable and as conscientious as themselves, to ignore the perplexity of the problem, and to regard with overweening confidence the result of their own inquiries. One scarcely ever stumbles on a new effort to remove the mystery hanging over this part of the heart's physiology, without having the memorable words of Turgot vividly recalled to the mind:—"Moins on sait, moins on doute, moins on a découvert, moins on voit ce qui reste à découvrir . . . . Quand les hommes sont ignorans, il est aisé de tout savoir."

128. Were we to address the mass of practical physicians, at home and abroad, who are untrammelled by the instincts of paternity in regard of any of the theories proposed, and who have besides directed serious attention to the study of heart-diseases, and ask them—does any single explanation of the cardiac sounds heretofore adduced appear to you to solve the problem?—the all but unanimous reply would, I feel no shadow of a doubt, be—No. For this reason, I think, it would prove a useless, as it most certainly would be a tiresome, task to review the group of theories *seriatim*. The better plan seems to be to enumerate with as much brevity as possible the phenomena which are as well coincident with the sounds as by possibility soniferous, and endeavor to ascertain, on such acoustic, anatomical, experimental, and clinical data, as we can command, which among the number actually concur in producing the sounds.

129. *Mechanism of the first sound.*—The phenomena which are at once conceivably soniferous and coincident with the heart's first sound may be arranged in the following manner:—

130. (*a*.) *At and immediately after the commencement of the sound.*—At this period occur (1) the sudden tension of the auriculo-ventricular valves; the sharp collision of certain portions of the auricular surfaces of those valves; the impulsion of some of the blood in the ventricles against the ventricular surfaces of those valves: (2) The attrition of the blood-elements *inter se* within the ventricles, and their impulsion with attrition upon and among certain portions of the columnæ carneæ: (3) The impingement of the blood, projected from the ventricles, against the orifices of the pulmonary artery and aorta, and perhaps against the bases of the columns of blood contained within those vessels, with the simultaneous flattening of the sigmoid valves against the arterial walls, and the diastolic extension

of those walls: (4) The impulse of the heart's apex against the side, or against lung-substance, if this be interposed: and (5) The attrition of the pericardial surfaces accompanying systolic movement.

(*b.*) *Towards the close of the sound.*—The collision of the surfaces of the ventricles, after the expulsion of their contained blood.

(*c.*) *Throughout the entire duration* of the sound, the sustained muscular contraction of the walls of the ventricles, together with, as a specific additional element of sonorousness, the perfect, completed tension of the muscular fibres.

131. Before considering the claims of these different agencies as productive of the first sound, let me remind the student of a very obvious truth commonly ignored or forgotten by writers on this question—namely, that sound or noise may by possibility be caused at the period of systole by many actions which, nevertheless, take no part in producing *the* first sound. It might be, again, that such superadded actions produced sounds, which sounds were habitually audible at the præcordial surface, or, on the contrary, either constantly or occasionally drowned *in transitu* thereto, either by the superior intensity of the true first sound itself or by interfering vibrations.

This premised, let us ask, first, which of all these possible elements of sonorousness actually prove soniferous—and, secondly, which of them contribute to form *the* first sound of the heart. The order in which we examine them is not of much importance.

132. The amount of noise, produced by the impulsion of the heart against the chest and its friction against the parietal pericardium during systolic locomotion, is too slight in the normal state of things to be audible to the listener through the chest-wall. Let, however, the action of the heart be simply abnormally increased in intensity, all other conditions remaining the same, and both these phenomena become perceptibly sonorous, each in its special way—the former as a knocking and somewhat toned noise against the side, the latter as a rubbing murmur. Both these facts are almost daily exemplified in clinical practice: patients with hearts excited by simple neurosis, or with hearts somewhat enlarged and dynamically excited besides, very frequently present a pericardial rub of single systolic rhythm which disappears under rest and treatment.[1] And even where there is no statical or functional disturbance of the heart, a knocking sound may in some individuals be heard with systole, especially where the posture of the trunk, the period of the respiratory act, or other conditions, extrinsic to the heart itself, allow the surface of the organ to play with unwonted vigor against the parietes. Still the practised ear may always succeed in recognizing that this sound is not *the* sound of systole—but that it is a something superadded and independent, differing in site, in quality, and sometimes in precise rhythm from this.[2]

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[1] *e. g.* Roberts. U. C. H., Females, Oct. 10, 1850.

[2] Where a thick layer of emphysematous lung intervenes between the heart and

133. There is a deficiency of evidence to show, that under the peculiar conditions of ordinary systolic contraction, the collision within the ventricles of the blood-elements among themselves is habitually sonorous: and, at all events, it may be assumed that any noise thus produced is inaudible on the outer surface of the chest.

134. Nor do I think proof has ever been given that the collision of the internal surfaces of the ventricles is attended with appreciable noise—much less that such collision normally contributes to the formation of the systolic sound, as is supposed. The hypothesis seems in truth contradicted at once by the fact that the blood of the ventricles is not completely expelled by their systole. It has been suggested that a clicking quality in the first sound, sometimes to be detected after profuse hemorrhage, may thus be generated: the idea seems to me more ingenious than judicious—and, even conceding its justness, it throws no light on the mechanism of the normal first sound.

135. Having thus eliminated certain of the attendant conditions of systole as sources of sound, at least of sound audible at the surface of the chest, which of the remaining phenomena may we fairly admit as taking part in the first sound?

136. Now that the systolic sound derives its special full, booming quality, in a great measure, by consequence, from the act of muscular contraction has, since the day of Laennec, been commonly accepted as a recognized fact. For, on the one hand, the sound has been found to be affected, as regards these characters, when the heart contracts after separation from the body and the action of the auricular valves is prevented; and, on the other hand, a very close imitation of the systolic sound is to be found in that of a powerful voluntary muscle strongly contracting.[1]

137. But only a share, not a perfect imitation, of that sound. There is another most important sonorous agency at work—the abrupt vibration of the stretched auricular valves, aided by their surface-collision at their free borders, coupled with scant shock of blood against their ventricular surfaces. This it is which impresses the special twanging character on the sound—a character which may be detected by attention at its outset, and which in certain states of altered contractility of the muscular fibres, almost covers

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chest wall, and sometimes even without this, I think there is reason to believe the first sound may be given an intermittently murmur-like character at the apex, independently of any disease in the mitral valve, simply by the apex-point of the heart impinging against the lung, and moving some of its contained air. I have observed the phenomenon (even during temporary suspension of the respiration), where this seemed its sole plausible explanation.

[1] In a case, unique as far as my experience and reading extend (seen with Mr. Hardwicke), of hypertrophy of the recti abdominis, attended with powerful contractions, partly reflex, partly consensual, of their tissue, the variation in intensity of the muscular sound, according as the contractions were slow and gradual, or quick and abrupt, was very striking: in the latter case, the sound bore, to my ear, a very distinct resemblance to the first sound of the heart.

or rivals in strength the sound generated by these: the elements of sonorousness of individual quality exist in both these conditions; and, when either is experimentally interfered with, a corresponding change is found to follow in the character of the first sound.

138. Whether, as I believe to have been particularly maintained by Dr. Hope, the walls of the ventricles become at a certain period of systole at once tense and vibrating, after the fashion of a stretched membrane, and so produce a specific addition to the muscular sound of contraction, has not been positively determined. The theory, however, seems acoustically as well as physiologically of questionable justness, and may, I think, be with safety ignored.

139. Further, the projection of the ventricular blood against the orifices of the large vessels, against the flattened sigmoid valves, and likewise probably against the bases of the columns of blood those vessels contain, combined with the sudden extension of the arterial coats beyond, have strong clinical and experimental claims to a share in *the* first sound. That the first of these phenomena is sonorous cannot be disputed: a sound is audible in the arteries, synchronously with the heart's systole, under circumstances in which the idea of mere conduction from the heart is quite inadmissible; such sound may be heard in the femoral and even popliteal arteries sometimes, where no disease of these vessels or of the aorta exists. Again, in certain cases of mitral regurgitant disease, where true systolic sound at the left apex is completely deficient (a murmur only existing there), the first sound may be discovered with much of the quality of health at the base. Still, however, it seems to me that the sound in question can only be viewed in the light of an accessory and not an essential element of *the* systolic sound.

140. In fine, it would appear that the *essential* causes of *the* first sound may be reduced to:—

Muscular action—that of the contracting ventricular walls;[1]
Sudden membranous tension—that of the auricular valves.

Then as a *constant* accessory cause of noise with systolic time must be admitted,

Forcible shock of fluid against resisting membrane—that of the blood against the orifices of the large vessels.

And, lastly, as *occasional* accessory causes of noise with systolic time rank,

Impulsion of solid against solid—that of the heart's apex against the chest-wall.
Abrupt friction of surfaces—that of the two layers of the pericardium.

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[1] The recent ingeniously conceived experiment of Dr. Halford, designed to prove that valvular vibration is the sole cause of the first sound, seems to me to alter the physiological condition of things so seriously, that the inference it appears to furnish, namely, that muscular contraction is non-soniferous, cannot be accepted. *Per contra*, M. Malherbe (Journ. de Physiologie; de Brown-Séquard, vol. i.) loses himself in a maze of difficulty in his attempt to prove that the ventricular valves cannot produce any sound at all.

But, though an occasional cause of sound [illegible] the impulsion of the heart against the chest-wall is not [illegible] of the first sound, as taught by Magendie, [illegible] when soniferous, actually either partially cover [illegible] of interfering vibrations, weaken the true first sound.

141. *Mechanism of the second sound.*—The [illegible] phenomena, synchronous with the second sound, are [illegible] of the ventricles, and rush of blood into their cavities; the sudden recedence of the heart's apex from the chest-walls; the [illegible] of the auriculo-ventricular valves to the sides of the [illegible] the sudden tension of the sigmoid valves, and impulse [illegible] column of blood against them during the arterial [illegible] systole itself.

142. Now, powerful though the diastole of the ventricles [illegible] even in the new-born infant to open [illegible] around them) it seems certain, as [illegible] on living animals, that the phenomenon is soundless. [illegible] as to the non-production [illegible] of blood into the ventricles. Both occurrences may be [illegible] non-soniferous in the normal state.

143. [illegible] the sudden partial separation [illegible] pericardium, which had come together in systole, [illegible] it would be difficult enough to prove [illegible] sound is so slight as to be inappreciable. [illegible] In disease the case may be different [illegible]

144. [illegible] experiments tending to eliminate [illegible] of the auriculo-ventricular [illegible] rhythm. There is certainly [illegible] second sound is in any measure thus [illegible]

145. [illegible] that the essential cause of the [illegible] taught from clinical observa[illegible] of the sigmoid valves: the [illegible] second sound at the aortic [illegible] orifice, in cases of in[illegible] sufficient proof of the fact. [illegible] its maximum force, as [illegible] its membranous origin, [illegible] great vessels.[3] Besides,

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[illegible] —Gazette Médicale;

[illegible] mechanism of the second, [illegible] established: the mat[illegible] experiments in which [illegible] statement is made, [illegible] by a prolonged [illegible] as to whether [illegible] at the base only the fall of the columns of blood on the arterial surface of the valves must reinforce the sound of valvular tension—although for obvious reasons that fall cannot be so sonorous as if the valves, instead of being opened out by the receding blood, as they are, were *first* expanded, and *then* received the shock of the fluid from above, as is affirmed to be actually the case by M. Hamernjk, who but revives an exploded opinion of Lower.

146. Whether the arterial systole itself contribute or not to the generation of *the* second sound must be admitted to be yet open to inquiry. But that the phenomenon is soniferous cannot reasonably be doubted; the occurrence of double sound in arteries, far distant from the heart, seems to me to settle the point.

147. Although diastolic jogging action and quasi-impulse may occur without actual disease of the heart, yet there is no proof of such impulse being *per se* soniferous; the more so as, even in cases of strong morbid diastolic impulse, that impulse does not distinctly produce sound. M. Magendie's idea that the normal second sound is produced by the shock of the right ventricle against the sternum takes rank among the unquestionable errors of its day.

148. Hence the sudden tension of the sigmoid valves, and the fall of blood upon them, seem in the *normal* state the sole demonstrated causes of the second sound.

149. It is altogether another question whether, in various states of disease, diastolic occurrences, naturally soundless, become sonorous, and so produce a second sound of their own—or whether certain adventitious phenomena may in those diseased states produce an imitation more or less close of natural diastolic sound. Concerning this matter Skoda argues, that as exceptional cases occur in which the second sound is weak at the base, and loud at the apex, while there is no diastolic impulse against the chest wall to afford a possible explanation, the sound must originate in part in the ventricular region. He suggests that it may be produced either by, first, the stroke of the blood under special circumstances against the ventricular walls during the diastole of the heart: or, secondly, by separation of the heart's apex from the surface, against which it had been pressed during the systole; or, thirdly, by separation from the chest-wall of the portion of pericardium which had been driven against this during the systole.

As for the first suggestion: we have admitted that in the normal state, the blood enters the ventricles from the auricles, with a current so calm as to prevent audible sound from being thereby produced in the former cavities. But in cases of highly marked aortic regurgitation, blood falls with notable force into the left ventricle,

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of the heart, or over its entire surface. From several passages in the context, the former seems the more likely; and hence the records at least of these experiments leave it still an open question, whether or not the natural second sound is normally, in any small degree, ventricular in site, or capable of becoming so, when aortic regurgitation exists.

and may conceivably generate sound. I have unquestionably heard, at the left apex, a distinct sound in more than one such case, while at the aortic base the ordinary regurgitant murmur alone existed: cases of the kind would probably be more frequently met with, were it not for the loudness, and transmission to the apex, of the murmur involved at the base.[1] I presume this intensified second sound cannot be the transmitted sound of the pulmonary valves, because I have found it stronger at the left than the right apex;[2] though it might certainly be argued that this greater intensity of diastolic sound at the left, might depend on greater intensity of diastolic murmur at the right, apex (murmur drowning sound): for, as I shall hereafter show, the rule in aortic regurgitant disease is, that the attendant murmur is better conducted down the sternum than over the left ventricular region.

The second and third suggestions are certainly plausible. And for this reason: if, while the ear is applied to a stethoscope on the chest-surface, a neighboring spot be percussed with the point of a finger, which is instantly withdrawn when the blow has been given, two sounds will be heard for each blow; the first strong, corresponding, of course, to the direct impulse; the second, very weak, to the removal of the finger from the point percussed. Now the recedence of the heart's point from the side is here imitated.[3]

In all this the usual ingenuity of Skoda is apparent; but he appears to me to have lent his influence too much to establish the erroneous conclusion, that because in certain states of disease the two last phenomena may produce noise of diastolic rhythm, *ergo*, they take part in generating the natural second sound. Some further facts, bearing on the subject, will be noticed in the account of reduplication of heart-sounds [176].

(II.) MODIFICATIONS OF THE HEART'S SOUNDS, OCCURRING INDEPENDENTLY OF CARDIAC DISEASE.

150. (*a.*) *Modifications occurring at the site of production.*—The description now given of the sounds of the heart applies especially to the male adult. The first sound is somewhat higher pitched and more ringing in females and in children than in males, and hence apparently more intense. In women, too, it is habitually more extensively audible than in men; but rather, I think, from the acoustic conditions of the chest generally than as a direct attribute of the sound itself.

151. Variation of attitude has little, if any, effect on the second sound: but the first is commonly stronger in the erect than in the

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[1] Flood, U. C. H., Males, vol. vii. p. 265.

[2] In a certain proportion of cases of mitral regurgitation with dilated hypertrophy, an intensified second sound may be found in the left apex;—its explanation will be sought hereafter.

[3] The result is still more satisfactory if the inner surface of the chest-wall be percussed in the dead subject.

reclining, and in the prone than in the supine, postures: the reasons are obvious.

152. The extent and direction of transmission of the natural sounds of the heart are modified by various changes in the conducting faculty of the structures and organs within, or on the confines of, the thorax; hence the obvious inference that, unless the absence of such changes has been established, we are not justified in inferring that a plus or minus extent of propagation depends on the state of the heart itself. Thus, as a ready illustration, enlargement upwards and inwards of the liver or spleen renders the sounds unduly audible in one or other hypochondrium; but alterations in the density of the lungs practically furnish the most striking and significant examples: these are treated of elsewhere.

153. When the heart is displaced by diseases extraneous to itself, it might be supposed that the points of maximum force of its sounds would likewise be displaced: this is true, however, to a much greater extent of the first sound than of the second, and for the evident reason that the apex of the organ is more movable than its base. In left pleuritic effusion, for example, when the maximum point of the first sound is carried to near the right nipple (say seven or eight inches out of its place), the maximum of the second scarcely swerves farther than to the right edge of the sternum, or one and a half inches from its post. Now it follows from this, that the line connecting the maxima points of the two sounds deviates, more or less extensively, from the almost vertical bearing it presents in health.

154. The intensity of the heart's sounds, and even the pitch of the first, is heightened by nervous excitement of all kinds: in the hysterical and epileptic paroxysm this is sometimes strikingly remarkable; emotion, whether of fear, anger, &c., has a similar effect, the sounds being in some instances audible even to the individual himself, and to by-standers at one or two feet distance from the chest. Diseases attended with marked debility weaken both sounds; but this effect is greatly more perceptible in the first than the second: the essential mechanism of the two sounds gives in each instance the clue to this difference.

155. Certain conditions change the combined force and quality of the first sound by influencing some one of its elements without affecting the others. In continued fever, the general weakness, impairing the muscular power, throws the valvular element into undue prominence, and gives the first sound a clicking character, akin to that of the normal second sound. Again, nervous excitement intensifies, and sometimes even gives a ringing, metallic quality to the systolic impulsive noise, produced by the projection of the organ against the side [132], so much so as to throw the real elements of the first sound into the shade. The second sound is comparatively little affected in either case.

156. (*b.*) *Modifications arising beyond the site of production.*—The changes, just noticed, occur at the spots of production of the sounds.

Can the heart's sounds be increased in force, or changed either in pitch or in quality, subsequent to their generation, and through the influence of conditions beyond the heart?

157. I have known both sounds materially louder in the site of a cavity under the left, more rarely the right, clavicle, than at the mid-sternal base. More than this, in a case of well-marked chronic consolidation of the apex of the right lung, *the second sound of the heart* (this organ, the arch of the aorta, and the pulmonary artery being, as far as ascertainable from signs and symptoms, perfectly healthy) *was decidedly louder under the right clavicle than at the mid-sternal base.* No such reinforcement existed at the sternal notch, nor under the left clavicle; the peculiarity was therefore not due to reinforcement in the arch of the aorta: and further, the mediastinum and left apex were, judging from their percussion-note, free from solid matter. Here, then, was a case where the second sound was louder at about three inches distance from its point of production than at the point itself: the pitch of the sound in the two places was identically the same.[1] The mechanism of increased tone was here that by unison-resonance.[2]

158. Although it be theoretically conceivable that fall or rise in pitch of the heart's sounds shall arise, independently of any other change, in the course of their conduction to chest-surface, I know nothing of such modification as matter of experience except under the circumstances referred to in the next paragraph.

159. The condition of the hollow organs of the abdomen, especially the stomach, sometimes curiously modifies the quality of the heart-sounds. When that viscus is distended with gas to a certain amount, the sounds resound within it with a high-pitched metallic ring, and so loudly, sometimes, as to be perceptible to, and seriously alarm, the patient.[3] Large cavities in the adjoining lung (especially if their walls be hard, and the pleural sac distended with gas) sometimes augment the heart's sounds by unison-resonance, perhaps by echo, in a similar way.

160. The number of sounds corresponding to each revolution of the heart may be increased, but not diminished, independently of structural disease in itself, in the manner to be presently described [174].

(III.) MORBID STATES DEPENDENT ON HEART-DISEASE.

161. These are referable to the heads of—I. Modified sounds; and, II. Adventitious sounds or murmurs.

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[1] In Allen, U. C. H., Males, vol. ix. p. 196, the same phenomenon was ascertained on the left side.

[2] Vid. Diseases of Lungs, 3d Am. edit., p. 134.

[3] Warren, U. C. H., Males, vol. viii. p. 35; aneurism of arch of aorta.

I.—MODIFIED HEART-SOUNDS.

162. The intensity or loudness, and the extent of transmission of the sounds, when modified by heart-disease, are in the direct ratio of each other.

163. (*a*.) *Increase* in both respects is observed to its maximum in dilated hypertrophy, the valves remaining sound; muscular substance exists in sufficient excess to intensify the muscular share of the first sound, while the thinness of the walls, as compared with the size of the whole organ and capacity of its cavities, favors resonance, and acts as a special source of loudness. The forcible propulsion of the blood into the aorta leads to proportionally forcible reaction of the arterial coats, and unusually sharp recoil of the column of the blood on the sigmoid valves—hence a loud, full-toned second sound. In simple dilatation, the first sound, louder than in health, is sharper and more toned: the heart is brought more extensively than natural into contact with the chest-walls, and the thinness of the muscular walls allows of short abrupt contraction. Induration of the walls of the heart weakens the muscular portion of the first sound, but intensifies the valvular; and, if the other conditions for this exist, gives peculiar intensity to the noise produced by impulsion of the apex against the side.

Functional excitement of the organ also intensifies the sounds, whether this be dependent on incipient inflammation of the organ itself or of its membranes, or on general febrile disturbance previous to the occurrence of debility. But nervous excitement is the most powerful intensifier of the heart's sounds; it will even temporarily produce a loud first sound from a pure solidly hypertrophous left ventricle, and taken alone has much more frequently been known to render the sounds audible at a distance from the surface than any organic affection of the heart. I doubt if the latter, unaided by nervous excitement, be competent to produce the phenomenon [154].

The quantity of blood circulating through the heart also modifies the intensity of the first sound. If the quantity be considerable, the struggle of the ventricle to force it on is considerable and prolonged; but the sound does not gain, indeed rather loses, in resonance and loudness. When the quantity is small, on the contrary, there is no such struggle; the ventricles contract abruptly and sonorously. It is under those circumstances that, as already mentioned [134], it has been suggested collision of the ventricular surfaces may intensify and modify the quality of the systolic sound.

164. (*b*.) The sounds are *weakened* where the muscular structure of the heart is encroached upon by morbid infiltrations, or by its own disease. Softening, obesity, fatty degeneration, cancerous infiltration, atrophy with fibrous infiltration, all impair its force, both in its muscular and valvular elements, for obvious reasons. In

dilatation with attenuation, the first sound, though sharp-toned, is feeble—probably from conjoined change in the sarcous structure.

In simple, and still more in concentric, hypertrophy of the left ventricle, the first sound is weakened at the apex in, it may almost be said, the direct ratio of the increase in mass of the muscular substance and decrease of the cavity. In extreme cases, the first sound may be completely deficient over the ventricle, and perceptible only at the base, or towards the ensiform cartilage. The compact packing of the muscular structure interferes probably with the freedom of vibration necessary for the generation of sound, and the diminished size of the cavity obviously impedes the play of its parietes. Even valvular action is diminished in scope, and while the mitral valve is of less proportional extent, it is often thicker than natural. The entire mass of the ventricle, too, does not contract simultaneously.

Accumulations in the pericardium of fluid, of air, or of air and fluid combined, weaken the sounds by removing their seats of production from the surface. I do not know clinically the effect of solid formations in the pericardium; they may be conceived to weaken the sounds in one way, and intensify them in another—severally in regard of production and of conduction.

Want of muscular and nervous tone, generally weakens the first sound, as well as modifies its quality.

In some rare cases that have fallen under my notice, while the first sound has proved so feeble as to be scarcely audible, the second has been full-toned, strong, and audible over the entire chest. The notion of a hypertrophous condition of the pulmonary artery suggests itself as a possible explanation; but I have no certainty of knowledge on the matter. In the cases referred to, none of the commonly-admitted affections of the heart or great vessels existed.

165. (*c.*) The *duration* of the first sound falls below the natural standard in dilatation without hypertrophy, or with attenuation, and under all circumstances which weaken the muscular, and throw into undue prominence the valvular, portion of the sound. The sound is lengthened more or less in hypertrophy, with moderate dilatation; and if, in addition, the mass of circulating blood be large, or if the aortic orifice be at all obstructed, the sound may be sufficiently prolonged to fill nearly two-thirds the period of each revolution of the heart.

The second sound is shortened by a thin paper-like state of the sigmoid valves and by thinness of the blood; its pitch rises under the same influences. The sound is lengthened on the contrary, by thickening of the valves, and a loose inelastic condition of their texture. Probably, too, thickening of the non-striated muscular portion of the arterial walls will lengthen their own systole, and hence, probably, the diastolic heart-sound. Another curious cause of prolongation of this sound will be by-and-by described.

166. (*d.*) The points of maximum intensity of the heart's sounds

are more liable to *displacement* by extrinsic than by intrinsic causes; the latter, however, in some forms influence them. Enlargements of the heart generally, whatever be their nature, *lower* the maximum point of the first sound. Simple, and *à fortiori* concentric, hypertrophies influence its position much less, in proportion to their mass, than the eccentric variety of the disease. Pericardial effusion, raising the apex, *raises* the point of maximum intensity of the first sound; and in some rare instances I have known this carried backwards, the sound being more distinct in the left vertebral groove than in the ordinary apex-region. Hypertrophy of the right ventricle lowers its own first sound; of the left, carries its own immediately to the left, as well as depresses it. In all cases of depression of the first sound, the second is similarly affected, but to a less degree. Marked eccentric hypertrophy of the auricles would also probably lower the first more than the second sound; but I do not know this from experience.

167. (*e.*) The *distance* of the heart's sounds from the ear of the auscultator is very manifestly *increased* in cases of fluid and gaseous accumulation in the pericardium. There is, however, a source of fallacy here; the sounds are commonly listened to in the natural situation of the apex, and not in the neighborhood of the nipple or fourth rib, whither that apex has been carried by the disease. Agglutination of the pericardial surfaces, on the contrary, brings the sounds *nearer* than natural to the ear; if not precisely at their maximum points, beyond these. Enlargement of the heart, sufficient to push away the left lung, will have a similar effect. The first is more affected by these changes than the second sound.

168. (*f.*) The *quality*, and with this the *pitch*, of the heart's sounds is subject to serious modifications. The first becomes dull, muffled, toneless, and in some cases almost null, at the apex, where dense hypertrophy is conjoined with a thickened inelastic condition of the auricular valves. On the other hand, where the ventricular walls are thin and the valves natural, the first sound becomes more or less clear, flapping, or clicking, with raised pitch; if those walls be in a state of eccentric hypertrophy, and the valves somewhat thickened, the sound assumes a clanging character. At least, these statements are in accordance with the majority of results; but exceptions, explicable, perhaps, sometimes by the state of the heart's texture—in others inexplicable—pretty frequently occur. It would be difficult to describe or explain, for instance, the varieties of quality found in the first sound of soft, flabby, fatty hearts, with healthy, or nearly healthy, valves. Elevation of pitch depends, as a rule, either on thinness of the muscular walls, or on predominance of the valvular element of the sound.

The first sound sometimes possesses a peculiar full-toned quality, without the least sharpness, while it is strongly accentuated at the commencement, and commonly prolonged: the nearest articulate symbol of the two sounds, under these circumstances, appears to be *b'oom-tup*, pronounced with strong emphasis on the *b*. I have observed this peculiarity in cases of eccentric hypertrophy of the left ventricle—but without ascertaining the special condition on which it depends.

The quality of the systolic sound at the apex seems sometimes sharply knocking; but with care this knocking quality is separated by the ear from the true heart-sound, and obviously depends on the impulse of the apex against the side—but not necessarily against the inferior border of the fifth rib, as imagined by Dr. Hope. Knocking impulsive sound cannot be called an essential or even habitual attendant on any particular disease of the heart; nervous palpitation, especially if the edge of the lung be by some disease of its own carried unduly towards the left, will readily produce it in a healthy organ. Thin-walled resonant chests supply it with greater ease than others; and morbid induration of the heart's apex, or calcification of the pericardium, will aid in intensifying it. The heaving and steadily pushing character of the impulse in simple hypertrophy prevents its occurrence in that disease: eccentric hypertrophy is the form of enlargement it most frequently accompanies.

169. The *first* sound is sometimes slightly rough, and approaching in quality to a murmur at the apex; it is, in fact, *murmur-like*, without being actually converted into a murmur.[1] This may be (*a*) a persistent condition, observable week after week, while the patient remains under treatment; or (*b*) a temporary state, constantly noticeable for a few days, and then disappearing; or (*c*) a mere transient phenomenon, occurring with some, absent from other, beats of the heart. In the first case (*a*), it has appeared to me referable to an incipient or slight amount of some one of the organic conditions which, carried further, produce a perfect systolic murmur; or, probably, sometimes to a buzzing murmur-like quality in the muscular sound itself, produced by slow contraction of the fibres, or some special alteration of their texture. The second case (*b*) is exemplified by some *excessively rare* instances of acute rheumatism, where the systolic sound at the left apex, roughened and murmur-like for a few days at the outset, then loses this quality, either permanently, or to resume it again at a later period in a more decided form.[2] Passing vascular roughness of the mitral, or even ventricular, endocardium, and imperfect closure of that valve from the influence of irritation, suggest themselves as possible causes of the phenomenon; but anatomical evidence is of course wanting on the point. In the third case (*c*), the peculiarity is caused either by coincidence of the respiratory sound with that of the heart,[3] or by

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[1] In the case already referred to [note, p, 60], when the contraction of the abdominal muscles occurred in a slow vermicular manner, the sound was distinctly of murmur-like quality.

[2] James Hayes, U. C. H., October, 1850.

[3] It is sometimes impossible to satisfy oneself on this point, unless by causing the patient to suspend his breathing.

rubbing of the apex against the pericardium,[1] or by movement of air in the adjoining lung-substance produced by the cardiac impulse, or (by far the most important cause, because the most likely to lead to error) by a tendency to reduplication [176] of the first sound.

170. The *second* sound at the base is rendered dull, and comparatively clanging, by fibro-fatty thickening, without insufficiency, of the sigmoid valves. Diminished elasticity of the arterial walls has a similar effect.

Like the first, the second sound may be murmur-like, temporarily or permanently. Very trifling insufficiency will probably thus modify its quality; whether marked reticulation of the valves will suffice for the purpose, will hereafter be discussed. The most common cause of murmur-like quality in this sound is a tendency to reduplication.

171. (*g.*) The natural *accentuation* of the sounds, as shown in a previous page, is liable to numerous perversions; but as the accent falls on whichever sound is intensified, and mainly at the spot of intensification, repetition may be saved by referring the reader to the paragraphs on augmented intensity. When the heart is weak and flabby from organic change, or from want of tone, there may be a total deficiency of accent on either sound at the apex;[2] the sounds resemble those of a vibrating pendulum.

171. (*h.*) Like the sounds, the *silences* vary in disease in relative duration. The first, or post-systolic, silence is lengthened by deficient elasticity of the arterial walls, whereby the recoil of the blood on the valves is sluggishly effected; so, too, whenever the first sound is disproportionately shortened, the first silence is lengthened. The first silence is normally so short, that it is difficult to appreciate its decrease.

The second, or post-diastolic, silence is lengthened in cases of advanced constriction of the mitral orifice; the process of filling the ventricle is laborious and slow, and hence the systole lags, as it were, behind its time. When the circulation is greatly slackened, the second silence is generally disproportionately prolonged.

172. (*i.*) The relationship of the sounds of the heart to the pulse varies in disease. In the normal state, the first sound is apparently synchronous with the diastole of the arch of the aorta, the pulmonary, carotid, and subclavian arteries; thenceforth, the further the vessel from the heart, the more distinct is the interval between the systolic sound of that organ and the arterial diastole. It is difficult to determine the possible length of interval consistent with health: but it may be affirmed, that if the diastole of the most distant vessels, as the posterior tibial and dorsal artery of the foot, is so much retarded as to become synchronous with the second sound, the state

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[1] The more superficial character and the influence of change of posture will generally distinguish this variety from a true murmur.

[2] The second sound is, in certain rare cases of mitral regurgitation, so intensified that the accent falls on it, even when ausculted at the apex.

is morbid. This retardation, which was first noticed by Dr. Henderson as an attendant on insufficiency of the aortic valves, may with care be detected in many, but unquestionably not in all, cases of that disease. Possibly where no morbid retardation can be discovered, the failure may depend, not on its absence, but on its being carried to such extremes, that the arterial pulse produced by one cardiac systole is nearly synchronous with the next. The only fact, however, I know of, supporting this idea, is, that it is in *extreme* cases of aortic regurgitation the pulse seems most disposed to stand in normal relationship of time to the heart's systole. The same sign exists in attenuated dilatation of the left ventricle also. Again, in health, the frequency of the pulse and the length of the systolic sound vary inversely as each other: a frequent pulse is the index of a short first sound, and *vice versâ*. The same relationship holds good in some morbid states; for example, in anæmia and in the reaction after hemorrhage. It is, on the other hand, occasionally perverted; the pulse may be infrequent, and the systolic sound short: in fatty degeneration, and in simple flabby softening of the heart, this perversion may sometimes be noticed.

The sounds of the heart are sometimes suspended for the precise length of time occupied by an ordinary revolution of the organ: they are said then to *intermit*. Not uncommonly such intermission recurs with considerable regularity; that is, after a fixed number of regular beats. Sometimes the systolic sound seems to *anticipate*, sometimes, on the contrary, to *hesitate* at, the proper moment of its occurrence—changes of rhythm closely connected with shortening or prolongation of the second silence. Sometimes a series of feeble and rapidly succeeding sounds follows others comparatively loud, slow, and deliberate; and there may be a certain uniformity in the number of each kind, and in the periods of their recurrence. Or the *irregularity* of the sounds may be complete, both in intensity and in rhythm, no two revolutions corresponding to each other in either character: there ceases to be any semblance of order in disorder. This excessive perversion exists in highly marked mitral contraction and regurgitation, in extreme softening, fatty infiltration, acute destruction of a portion of a valve, or of chordæ tendineæ, rupture of these structures, formation of fibrinous coagula within the heart, and in a small proportion of cases of pericardiac effusion.

173. The natural correspondence in the number and time of cardiac systoles and arterial pulsations is habitually maintained, even when the rhythm of the heart's contractions is thus variously altered. If the left ventricle intermits, or anticipates, or hesitates, or becomes wholly irregular in its contractions, a precisely similar change occurs in the arterial pulses: the impulses of the connected tubes are the counterparts of those of the central organ. But, on the other hand, there may be a failure of this correspondence, not only when the heart's contractions are thus abnormal in rhythm, but even

when they are in this respect normal. Thus two revolutions of the heart may correspond to a single radial pulse, the cardiac action and the pulse being perfectly regular in force and rhythm; or the pulse may be perfectly regular, and the heart's successive systoles somewhat unequal in force and duration, as in a case formerly recorded,[1] where eighty-eight systolic contractions produced forty-four radial pulsations. Here the rhythm of each pair of beats of the heart might be represented thus: Systole = 5, diastole = 3; systole = 9, diastole = 4. It was the first of the two systoles that failed to affect the pulse at the wrist; and as there was no evidence of aortic or mitral disease, but merely of flabby enlargement, that systole may have been simply too weak to influence the distant vessels: the state was of temporary duration. I have observed a similar condition, persistent, but of less regular type, in cases of extreme contraction of the mitral orifice: under these circumstances, doubtless the systole occasionally takes place before the ventricle is supplied with blood to propel.

Again, in certain cases of utter irregularity of the sounds there may be no traceable accordance between them and the force or rhythm of the pulse. This is, perhaps, best observable where the irregularity comes on suddenly from rupture of a valve, or accumulation of coagula in the cavities; but is occasionally met with in all the diseased states productive of irregularity.

174. (*k.*) The *number of sounds* attending each beat of the heart may vary, the arterial pulse holding its natural relationship to the systole.

175. A *single* sound only may be heard, and this may be the first or the second; whichever sound be deficient in any particular spot, it may, or may not, be audible at some other part of the cardiac region. The first sound may be quasi-deficient at the left apex, when the conditions, already described as weakening it, are carried to extremes; but it will then be found at the right apex, and at the base. So, again, the second sound may be quasi-deficient at the base from excessive feebleness, or from being covered by a prolonged systolic sound, or systolic murmur: but in the first case, excitement of the heart, increasing the energy of its contractions, will invigorate the sound, and in the second case, the sound will be heard at the right apex. Absolute deficiency of either sound, or of a murmur taking its place, has never fallen under my observation; in other words, neither systole nor diastole has ever been, in my experience, absolutely noiseless over the entire cardiac region.

176. *Reduplication of cardiac sounds.*—The systolic or diastolic sound, or both of them, instead of being single, may be double; the affected sound is then said to be cleft, doubled, or reduplicated. The phenomenon is far from uncommon in some of its forms.

Such slight accentuation as exists falls usually on the first of the three or four sounds: the pitch of the two divisions of the doubled sound is sometimes distinctly different. Reduplication existing at one spot may be audible elsewhere, or limited to that spot. The forms of reduplication, which have fallen under my notice, are the following:—

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[1] Clinical Lectures, Lancet, 1849, vol. i. p. 443.

1. *First sound.*—Double at the left apex (*ŭbbŭp-dŭp*), at the same time single or double at the right apex, and single,[1] or, more commonly, double, at the base.

2. *First sound.*—Double at the right apex (*ŭppŭp-tŭp*), single at the left apex and at the base.

3. *First sound.*—Perfectly doubled at the base, imperfectly so at the tricuspid apex, single at the mitral apex.[2]

4. *First sound.*—Double at the base, or at the apex, while the second sound is conversely double at the apex or the base.

5. *First sound.*—Double at the left apex, single at the base; two days later the doubling inaudible at the base and at the left apex, but distinct at the right apex, or upper edge of the ensiform cartilage.[3]

6. *Second sound.*—Double at the base (the most common of the series), very generally single at the apex: the reduplication audible or inaudible at the pulmonary and aortic cartilages both, or audible at either singly.

7. *Second sound.*—Constantly double at the tricuspid apex, and single at the base and left apex;[4] or, while constantly double at the tricuspid apex, occasionally so at the base.[5]

8. *Second sound.*—Constantly double, beat after beat, at the mitral apex; reduplication less distinct at the tricuspid apex; imperfectly or not at all reduplicate at base, and always attended with faint murmur (the difference in the amount of reduplication at the base and mitral apex is very striking); slightly reduplicate at pulmonary, not at aortic cartilage.[6]

9. *First and second sounds.*—Both reduplicate (*ŭppŭp-tŭrrŭp*). This is very rare, is more frequent at the left apex than elsewhere, and has never appeared to me audible at all parts of the heart's area.

There are, besides, occasionally observable curious modifications of the phenomenon of which the following are perhaps the most remarkable. Reduplication may change from one sound to another.[7] Doubling of the second sound at the base may be arrested by deep inspiration; or the second sound at the pulmonary cartilage, single in expiration, may be most distinctly split into two at the end of inspiration.[8] Sometimes, again, a tendency to reduplication exists without the sound being actually double; this state of things prolongs, and may give a murmur-like quality to, the affected sound (169).

177. The essential *cause* of these various reduplications seems to be a want of synchronism between the actions of the two sides of

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[1] Bowry, U. C. H., Males, vol. vii. p. 259.

[2] Branchley, U. C. H., Females, vol. ix. p. 94.

[3] Lenton, U. C. H., Males, vol. x. p. 86.

[4] J. Hayes, U. C. H., Males. Oct. 17, 1850.

[5] W. Hodson, U. C. H., Males, vol. ix. pp. 65, 66.

[6] Couch, U. C. H., Females, vol. vii. p. 343. The above true, as far as the words "at base," of Dunn, U. C. H., Females, vol. xvi. p. 296.

[7] Branchley, U. C. H., Females, vol. ix. p. 146. "At base systolic sound doubled, when first listened to; within two minutes the second reduplicated: no doubling of either sound at left apex during examination."

[8] S. Roberts, U. C. H., Females, vol. v. p. 220.

the heart. If the facility with which the two ventricles fill with blood be unequal, they will probably differ somewhat in their time of contraction; certain conditions of the auriculo-ventricular valves may bring their closure on either side, as it were, behind time; the production of a double systolic sound at the apex becomes thus readily intelligible. But the fact that such reduplication may be audible at one apex only, and perfectly inaudible at the base, is far from being easily explicable; it cannot, except on the merest hypothesis, be referred to double spasmodic contraction of either ventricle, and in point of fact did such contraction occur it would be attended in great probability with double systolic impulse, and this I have failed to detect in cases of the sort. It appears, too, from a case observed by M. Charcelay,[1] that the contraction of the auricles, when highly hypertrophous, may become sonorous, and so double the first sound; but it is scarcely necessary to add, that such mechanism is of singular rarity; nor does it appear probable that the characters of the reduplicate sound could, under the circumstances, resemble those ordinarily met with.[2] A simulated reduplication of the first sound may sometimes be produced by the knock of the heart against the side; but the least practised ear will readily distinguish this from true *intracardiac* reduplication.

Non-synchronous tightening of the sigmoid valves, again, easily explains the reduplication of the second sound at the base; and may itself be referred to unequal elasticity in the coats of the aorta and pulmonary artery, stiffness of either set of valves, a material obstruction in the way of their closure, or any state of either ventricle rendering it slightly tardy in the propulsion of its blood into the artery beyond. The fact that inspiration, especially if full,[3] will sometimes cause doubling of the second sound, which is inaudible in ordinary breathing, may be explained by the unduly abrupt rush of blood into the pulmonary artery, whereby the necessity for closure of its valves, to meet the recoiling fluid, is felt a little earlier than usual. But the same difficulty, as in the case of reduplicate systolic sound, arises in accounting for the limitation of the phenomenon to certain points of the cardiac region.

178. *Diagnostic Signification.*—In regard of diagnosis, it must be confessed, these reduplications are almost insignificant in the present state of knowledge. And for the following reasons: reduplication is never, as far as I have observed, permanent and invariable; it occurs most commonly in hearts either healthy or temporarily disordered in function only: less commonly in cases of slight organic affection; and with least frequency when serious valvular

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[1] Archives Générales de Médecine, 1838, p. 393.

[2] I have known a hypertrophous left auricle produce a distinct, even loud *knocking* sound at the third left cartilage.—Fred. Smith, U. C. H., Males, vol. v. pp. 297–8, Nov. 1850.

[3] Shillingford, U. C. H., Females, vol. xiii. p. 110.

disease exists;[1] it is not connected, as a rule, with any particular form of disease, either of the heart itself, or of the system, rheumatic or other; it comes and goes in the course of a few beats of the heart; sometimes disappears on change of posture, and may be affected, nay, induced, by the act of respiration.

179. The real interest of reduplications arises out of their bearing on the theory of the heart's sounds—a fact of which the following illustrations afford sufficient evidence.

The second sound may be continuously doubled at the base, and perfectly pure and single at the apex. How is this explicable on the simple sigmoid theory of the second sound? A double sound does not become single by conduction over so short a space.

The first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex. Here the true musculo-valvular first sound seems to be separated at the right side of the heart from the incidental element of systolic sonorousness, caused by impulse of the blood against the orifices of the great vessels [139].[2]

The second sound may be double at the base, and single at the aortic, double at the pulmonary, cartilage, or *vice versâ*. This cannot arise from want of synchronism of the two sets of valves, but of the three divisions of one set.

The second sound may be single at the base and double at the left apex; now, according to the pure sigmoid theory, the arterial valves are the sole source of the second sound: how come the two sets to divide their compound sound at the apex? Splitting into two, as a result of conduction from the base to the apex by ventricles of different conducting powers, cannot be admitted; for the reduplication may be present at the left apex only, absent at the right. This is the strongest fact I know of, in favor of the second sound being in some cases partly of ventricular origin [149].

II.—ADVENTITIOUS SOUNDS, OR MURMURS.

180. Sounds of adventitious origin and properties, produced either within or on the surface of the heart, are conventionally termed murmurs. In applying the term to any sound heard in the cardiac region, we imply that the precise mechanism and the intrinsic properties of that sound differ from those of the true heart-sounds.

181. In reference to their seat of production, cardiac murmurs are divided into endocardial and pericardial.

A.—ENDOCARDIAL MURMURS.

182. The *special character* of all endocardial murmurs is, more or less, blowing. Their quality varies extremely, and they may be

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[1] It is fair to observe, however, that the existence of murmurs in such cases may be the real cause of reduplication not occurring, or not being heard.

[2] In one case (James Hayes, U. C. H., Oct. 17, 1850) where this form of reduplication was well marked, pericardial adhesions had recently formed.

called simply blowing, grating, filing, rasping, sawing, whistling, or cooing, according to their greater or less similarity to these sounds. The simple blowing murmur, though itself presenting different degrees of harshness, is always soft in comparison with the filing, grating, rasping, and sawing varieties. In some cases they are tones, capable of musical notation. Endocardial murmurs may become audible to the individual himself [302]; they may in some instances be heard at short distances from the chest.[1] They are essentially *intermittent;* and, no matter what its duration be in relation to the heart's contractions, a single murmur is never sustained continuously, either in a uniform or remittent manner, through a series of beats. In point of absolute duration, they vary from a scarcely appreciable moment to two or three seconds: the latter amount of prolongation can only occur where the physical conditions are at once peculiarly favorable for sustainment of sound, and the action of the heart extremely slow. Their *pitch* varies by several notes, the lowest being, perhaps, represented by the whispered word *who,* the highest by *ss;* intermediate notes may be represented by whispering the word *awe* by inspiration, and the letter *r,* with various degrees of closeness of the isthmus of the throat: these are the suggestions of Dr. Hope. In point of apparent *distance from the surface,* they vary also; so deep, in some cases, as obviously to be weakened by distance; in others, they seem to originate directly under the integuments. The spots of *maximum force* of individual murmurs, the position of the heart being normal, are four—a few lines above the left apex; just above the ensiform cartilage; at mid-sternum, on the level of the third interspace; and at the junction of the third left cartilage with the sternum. But these points of maximum force are liable to change, both from various malpositions of the heart, and from alteration in the conducting qualities of the materials around it; the laws of transmission are then the same as in the case of the healthy sounds: a special cause, the direction of the current of blood, will be considered hereafter. An endocardial murmur once developed is habitually *persistent,* and attends every beat of the heart; however, weak systoles may fail to produce a murmur well marked with strong ones; tendency to syncope, general debility, and collapse, and the approach of death may prevent its production; and sometimes (for instance when the cause of the murmur is pressure on an arterial orifice by a tumor) certain postures may annul a murmur completely. Murmurs habitually attain a higher type of harshness the longer they exist, though a fall in pitch often takes place.

183. The *rhythm* of murmurs may be considered in respect of the heart's contractions and of the heart's sounds. In respect of

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[1] I have only once to my recollection met with this. In a case of cyanosis occurring in a child about six years old, a systolic rough blowing murmur could be heard at a distance of an inch or a little more from the surface.

the former, they are said to be systolic and diastolic; in respect of the latter, synchronous with the first or second sounds. But they are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, or post-systolic; pre-diastolic, diastolic, or post-diastolic: they may occupy a portion only of either time; or fill this and the succeeding silence, and encroach upon the succeeding sound—as is common with systolic, rare with diastolic, murmurs. This arrangement is doubtless open to the charge of hyper-division; but as it positively has its foundation in correct clinical observation, it ought to be kept in view, although for ordinary purposes the simpler one is quite sufficient: there can be little doubt that many of the alleged failures of the rules for valvular diagnosis are traceable to inattention to these subdivisions of systolic and diastolic time. But murmurs are further divisible in respect of rhythm; whether systolic or diastolic they may occur in the direction of the current of blood, or against it. Thus the systole of the left ventricle may produce a murmur at the aortic orifice with the current, or at the mitral orifice against it: hence a division of murmurs into direct and indirect—which are also severally called, from their common causes, murmurs from constriction and from insufficiency of valves, or from obstruction or regurgitation through orifices.

184. The effect of murmurs on the sounds varies. A murmur may simply render a synchronous heart-sound obscure, at its commencement, at its close, or throughout its entire duration; or it may completely mask this by its intensity, and even similarly affect the succeeding sound; or it may prevent the natural heart-sound from being formed. Thus at the left apex a systolic murmur may completely drown a systolic sound, which is readily audible at the right apex and at the base. The systolic sound is not masked there, but really deficient, when both auricular orifices are not in a position to produce natural sound, but murmur alone, and the arterial, or basic, portion of the first sound is feeble, or itself converted into a murmur.

When, as is very common, both murmur and sound are distinguishable coetaneously, the state may be called one of pseudo-reduplication. But true reduplication of a murmur is so excessively rare, that I remember to have met with but two examples of the fact: one, basic and diastolic, must have been in the aorta alone, for the pulmonary artery was unaffected: of the other, systolic at the left apex, I had no opportunity of examining the mechanism;—it might have been mitral and tricuspid together, or it might have depended on coexisting deep-seated venous hum—in which case, of course, it was a false reduplication only. I once heard a post-diastolic basic murmur and sound in a rheumatic woman, in whom the doubling appeared to arise in consequence of the set of sigmoid valves, which gave the murmur, acting after the set which gave the sound.

185. Some murmurs are unaffected, others affected more or less seriously, by the posture of the patient. A systolic murmur limited to the left apex, and highly marked in the sitting or erect, may totally disappear in the supine posture, instantly reappearing when the patient again sits up. Basic systolic anæmic murmur, will often cease in the erect, to return in the lying, posture. Systolic murmur, strongest at the left apex, and powerfully marked in recumbency, may disappear in the erect posture, remain inaudible for several beats after the patient has again laid down, and very gradually recover its previous intensity.[1]

Is this changeableness of a murmur proof of its inorganic nature? My experience positively supports the negative.

186. When endocardial murmurs have existed, structural changes, dynamic perversion, or morbid conditions of the blood are found to explain them, hence their division as follows:—

- (a) Organic.
- (b) Inorganic
  - Hæmic
    - Of changed composition.
    - Of coagulation.
  - Dynamic.

187. (a.) *Organic Endocardial murmurs* are essentially connected with such alterations of the orifices or of the valves, as, while they lead to constriction or imperfect closure of the orifices, cause unnatural friction between the blood and surfaces. The chief of these alterations are: simple *constriction*, or constriction with thickening, hardness, rigidity, calcification, warty or other excrescences from, or even simple inflammatory loss of polish and roughness of, the valves;—simple *insufficiency* of the valves to close a widened orifice, themselves not having grown *pari passu* with the widening, or such insufficiency depending on the various diseases of the valves just enumerated, or depending on shortening and thickening of the chordæ tendineæ or on atrophy or contraction of the columnæ carneæ, or on puckering of valves, or adhesion of the divisions of a valve *inter se*, or to the adjacent surface;—*excrescences* or other thickening or calcification of the valves, without either insufficiency or constriction;—*attachment of a tendinous cord* in a wrong situation, whereby it is thrown directly across the blood-current; or *unnatural communication* between the different compartments of the heart, or between these and the arteries or some adventitious cavity. Besides, without distinct alteration of the orifices or valves, mere vascular roughness of the ventricular endocardium probably suffices to affect the purity of the sounds, when the current bears, especially towards the arterial orifices, on such a roughened spot. Coagula among the columnæ, or a polypoid body hanging from the neighborhood of the valves, will have a similar effect.

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[1] Constable, U. C. H., Females, vol. ix. p. 96. Here the patient was spanæmic, the aortic valves slightly diseased, the mitral not obviously unsound—but whether regurgitation might or might not have occurred during life is matter of doubt.

Physically speaking, then, there appear pure *constrictions* of natural orifices, pure *widenings* of orifices, and pure *roughness* of surfaces, to explain the ordinary mechanism of organic cardiac murmurs. Now pure constriction and pure roughness are positively capable of producing murmur: this is matter of experimental demonstration; by pressing with the stethoscope on an artery, we convert the dull fillip-like natural sound of the vessel into a whiffing murmur. But how can pure widening of an orifice produce murmur? In the case of the tricuspid and mitral orifices, the *regurgitation* from such a physical cause may intelligibly produce murmur by the collision of direct and indirect blood-currents coming from and going back into the auricle: in the case of a *direct* current through a simply widened aortic orifice, the rippling of the stream, produced by the change of calibre, seems, from the experiments of Dr. Corrigan, sufficient, if that current be strong, to produce murmur.

188. Mere alteration in the direction of the current, of a kind to throw the blood obliquely against, instead of carrying it directly through, an orifice, will theoretically generate murmur. Probably this divergence of the stream plays a part in many valvular murmurs produced by the onward blood-current: and as dilatation of the ventricles renders them more spherical and less convergent to their arterial outlets, it has been urged by Dr. Blakiston and others, that such state must produce murmur if hypertrophy coexist—that murmur actually is heard frequently under the circumstances—and that, when wanting, its deficiency depends on the muscular energy being impaired by disease.[1] Hypertrophy with dilatation of a cavity, if its arterial outlet remain undilated, puts that outlet relatively in a state of coarctation: hence, too, may arise a murmur. I will return to these points with the subject of eccentric hypertrophy.

189. It was suggested by M. Martin-Solon that the pressure of the heart and great vessels by abundant pericardial effusion might cause murmur;—in a case of the kind, murmur, well marked in recumbency, disappeared when the patient stood up.[2]

190. The *properties of murmurs vary greatly with the conditions of the fundamental cause producing them*, and even with some conditions independent of this.

191. The force, loudness, or *intensity* of a murmur increases with the vicinity of its origin to the surface—the density and hardness of the heart itself, and of the textures lying between the heart and surface—the force and velocity of the current—the amount of narrowing at an orifice—and the volume of blood propelled through the obstruction. Excite a tranquil heart, and a murmur, previously almost inaudible, becomes distinct; weaken the energy of

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[1] Murmurs thus generated by *misdirection of the blood-current* would really belong to the dynamic species.

[2] Journal Hebdomadaire, ix. 457.

cardiac contraction by digitalis or aconite, and the converse result follows.[1]

192. The *quality* varies with the character of the surface over which the blood passes;—harsh and rough, if the surface be sharply uneven; soft, if smooth and merely constricted. But this influence is greatly modified, and may be actually reversed, by changes in the velocity of the circulation. The quality will also be materially affected by the condition of the intervening structures: if these be soft, the sound will be softened in quality; if hard, hardened. A musical quality is sometimes given when prominent spiculæ, of vibratile character, project into the current; and also when rigid vibratile edges bound a narrow chink-like opening.

193. The *duration* of murmurs will increase directly as the extent of surface in the condition to afford them, the amount of difficulty to struggle against, the quantity of blood, and the slowness of the circulation. If the structures intervening between the seat of murmur and the surface be imperfect conductors, the audible sound will be shortened—its termination will be lost through imperfect conduction.

194. The *pitch* of a murmur is more under the influence of the size of the orifice through which the sonorous stream passes, than anything else: the smaller the orifice, the higher the pitch. But it is also raised by the tension of the walls of that orifice, and the thinness of the blood. The velocity of the current does not influence it, Dr. Blakiston urges, unless, as in blowing with a flute, the harmonic be elicited. But unless the edges of the diseased orifice be of such rigidity as to simulate the embouchure of the flute, it appears to me the two cases are not comparable; and as matter of actual experience I think it will be found a slight, though sensible, rise of pitch frequently takes place in murmurs when the circulation is excited.

The mere distance of the site of production of murmurs from the surface can in theory have no direct influence on their pitch. The same physical and dynamic conditions will generate sounds of the same pitch, whether they be close to, or as far as possible from, the chest-wall.

But may the pitch of a murmur change in course of conduction—may the pitch differ, as perceived at different parts of the parietes of the chest? To whatever distance a note continues audible, it is commonly held, provided no intervening change of medium occur, to remain the same note, as at the spot of its generation.[2] However this be, it is certain that where any change of conducting medium occurs *in transitu*, a modification of pitch of the conducted sound will, or may, arise. If there be many such changes, occurring between the point of generation of the sound and the point where it is listened to, the varying effects of each on the pitch of the traversing sound, may by possibility correct each other in such manner, that the resultant at the end of the series does not perceptibly differ in pitch from the original tone.

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[1] In a woman now under observation (Emma Powell, U. C. H., Dec. 1850), the systoles are so unequal in force that while some give a strong radial pulse others produce none; the strong systoles are attended with systolic basic murmur, the weak ones with none.

[2] Some experiments lately made by Mr. S. Ringer have led him to the conclusion that alteration of pitch occurs in tones conducted through one and the same medium—that the ticking of a watch, for instance, rises in pitch the further it is

Now these propositions are, as matter of experience, supported by clinical observation of cardiac murmurs; indeed, it was the observation of particular facts connected with the heart that led me to look after evidence to elucidate the general law.

Theoretically, change of the pitch of every individual murmur may occur according to the nature of the intervening media between the spot of production and of audition. Practically, such change is not often to be substantiated. For reasons not readily assignable, the murmur of aortic regurgitation furnishes the most marked and most frequent examples. Thus it is very common to find that murmur of higher pitch at the ensiform cartilage than directly over its site of production, the base of the heart; it rises by conduction. But the very same murmur, ausculted at the left apex of the heart, may have a different pitch from that of the other two spots—lower somewhat than that of the neighborhood of the ensiform cartilage, yet higher than that of the basic region. Difference in the conducting media in the two directions must be the cause of the difference in tone.[1]

I do not think the manner or degree of alteration in pitch are sufficiently uniform in occurrence to afford trustworthy indications in diagnosis.

195. *Influence of palpitation on pre-existing murmurs.*—The influence of palpitation on already established murmurs varies. As a general rule, strong palpitation of regular rhythm intensifies the whole series—the regurgitant quite as markedly as the direct class. I have, on the other hand, occasionally known a murmur, when audible in a calm state of the circulation, disappear during palpitation—but this only in some cases of mitral regurgitation. Can its cessation depend on irregular contraction of the wall of the ventricle allowing of such slight and feeble regurgitation that morbid sound cannot be generated? Again, in a case of enlarged, soft, flabby heart, with a congested state of the lungs and abdominal viscera, so long as the heart's action was in its habitual state of frequency and irregularity of rhythm, no murmur could be detected

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heard in air from the point of its production, while with certain other kinds of medium a fall may take place.

[1] Flood, U. C. H., Males, vol. vii. p. 265, May, 1852. Case of highly marked aortic regurgitation: "the murmur tailed on to the second sound is higher in pitch at the ensiform cartilage than at the base . . . passing from the former point towards the left apex, it falls in pitch."

either in the erect or recumbent postures; but when the number of the pulse had been reduced by digitalis, a distinct murmur, systolic at the left apex, attended every beat of the heart.

196. From various of the considerations into which we have now entered it would follow that the properties of a murmur, as caught by the ear, are every one of them, singly, of complex mechanism—and herein appears an easy clue to the absolute failure of all clinical attempts to establish the *precise amount and character* of the organic changes in a set of valves from the consideration of any one property, such as roughness, of the murmur they generate. Every necessary organic condition of a harsh murmur may be present, and yet the resulting murmur be soft, if the current be feeble—nay, murmur may be wholly deficient.

197. (b.) *Inorganic Endocardial murmurs.*—Endocardial murmurs, that cannot be traced to any organic cause after death, or that disappear so completely and permanently during life, as to preclude the idea of structural change in their production, are termed inorganic. They are divisible into two sub-classes—murmurs originating in some unnatural condition of the blood (*hœmic*), or in perverted action of the heart (*dynamic*).

Hœmic Intra-cardiac Murmurs.

198. Murmurs originating within the heart and dependent on an unnatural state of the blood, are referable to two classes: in the one (α) the blood is altered in composition, but perfectly fluid; (β) in the other its composition is not demonstrably changed, but from some cause or other it has coagulated in the heart's cavities.

199. (α.) *Murmurs depending on changed composition.*—An intracardiac hæmic murmur of this variety is of moderate or very slight intensity, commonly of medium or low pitch, short or moderately prolonged, of whiffing quality, very easily rendered temporarily harsh by excitement of the heart, and modified in intensity by certain changes of posture. This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and of the pulmonary artery—with a force at each proportional to the power of its communicating ventricle; scarcely conducted along the aorta at all; frequently audible, on the contrary, at the second left, or pulmonary [19][1] cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex. The site and rhythm of hæmic murmurs, excluding all those of diastolic time and of seat at either apex, are of great value in their distinction. To their quality and pitch I attach but moderate importance; for organic murmurs may

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[1] This clinical fact does not appear to me to prove that anæmic murmur is more commonly generated at the pulmonary than at the aortic orifice, but simply that the nature of the media lying in the direction of the pulmonary cartilage is more favorable to conduction.

be soft, and inorganic ones are not very unfrequently rather harsh; while the latter may be shrill, whistling, and of high pitch, and the former are of course frequently of low pitch. Unfortunately there is no character in a systolic basic blood-murmur which positively proves its nature, and distinguishes it, *under all circumstances*, from one of organic source. The distinction is often rather to be made through the absence or presence of venous hum (I do not remember ever to have observed an intra-cardiac spanæmic murmur unattended with venous hum), and the course and duration of the phenomenon, than through its own intrinsic characters: *permanent* harshness and high pitch are never associated with true hæmic murmur. If arteries, on which the pressure of the stethoscope can have no influence, as, for example, the arch of the aorta, the innominate and subclavian arteries, be the seat, extensively, of strong blowing murmur, a co-existing systolic murmur at the mid-sternal base is, in part at least, inorganic; but, on the other hand, the cardiac murmur may be truly inorganic, and yet the arteries be perfectly free from abnormal sonorousness.

The morbid state of the blood most frequently associated with these murmurs is spanæmia—whether it be that of chlorosis, of malaria, of starvation, of deficient insolation, of hemorrhage and over-venesection, of carcinoma, of convalescence from acute diseases, &c. I have occasionally observed it in uræmia; so, again, it occasionally occurs, though to a very slight amount, in the hypinosis of continued fever and the exanthemata, and in the hyperinosis of pneumonia and acute rheumatism, under circumstances excluding more or less positively its dependence on endocarditis. It has been affirmed that plethora, rendering the quantity of blood too great for the cavities of the heart, produces murmur within the organ: confirmation of the statement is wanting; more especially if we accept the teaching of those chemists who maintain that the relative quantity of red corpuscles is increased in plethora.

The mechanism of this variety of hæmic murmurs will be considered with that of the venous class.

200. (β.) *Murmurs depending on coagulation.*—These murmurs form a link between the inorganic and organic classes. Their varieties are exceedingly numerous. Here must in strictness be included all murmurs depending on fibrinous deposits on the endocardial surfaces, wart-like, stratiform, or other; and on similar accumulations in fissures of the valves or tendinous cords, or between the columnæ carneæ. In these cases, however, the process of deposition is slow—the essential conditions involve, practically speaking, structural changes, and the attendant murmurs are unquestionably not to be clinically distinguished from those of ordinary organic mechanism. But to the present group belong also murmurs caused by sudden coagulation within the heart's cavities —and to these only is the following description to be understood to refer.

201. These coagulation-murmurs are materially most common on the right side of the heart, simply because the tendency of the blood on that side to coagulate during life is greater than on the left. Clots are obstructive to the onward current—these murmurs are consequently of direct mechanism, systolic when produced by clots in the ventricle. It is conceivable, that a murmur of this mechanism may be of præsystolic or diastolic rhythm also, when clots accumulate in the auricle, or at the tricuspid orifice in such manner as to obstruct the onward current, or at the arterial orifices in such manner as to cause regurgitation. But I have not actually observed any of these conditions. The pitch of these murmurs is low or medium, and, commonly, soft; they are never, as far as I know, markedly harsh.

202. The circumstances under which intra-cardiac coagulation occurs, and under which murmurs may be looked for, are mentioned in the account of BLOOD-CONCRETIONS WITHIN THE HEART.

Dynamic Intra-cardiac Murmurs.

203. Under this head, murmurs occurring in the heart through abnormal action range themselves.

204. (a.) Violent excitement of the organ, whether it act merely by increasing the force of the current, or by disturbing its direction, occasionally produces for the time systolic murmur at the base. I have observed this not only in hysterical females, who, though of florid countenance, might, it is true, have been slightly spanæmic, but in males with palpitating heart. If the organ be the subject of dilated hypertrophy, palpitation is sometimes, but certainly not always, attended with the same murmur. In hypertrophy with dilatation there is possibly, we have seen, another dynamic source of systolic murmur: from the altered form of the cavities of the ventricles, their contained blood is probably propelled against the edges of the arterial orifices, instead of directly through them; and this misdirection of the current may, very possibly, generate murmur [188].

205. (b.) The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class.

Thus, a systolic murmur, free from harshness and of medium pitch, having all the attributes of that denoting mitral regurgitation, is occasionally heard in cases of chorea at the left apex. This murmur disappears as the primary neurotic disease itself disappears; its intensity varies with different beats of the heart; its quality similarly changes; and it may even temporarily become inaudible. The murmur in question is not localized as a hæmic cardiac murmur would be [199]; it cannot be referred to organic change in the mitral valve, seeing that it eventually totally disappears. Nor in many of the cases which I have seen was there the smallest ground for referring it to latent Bright's disease, or rheumatic irritation of the endocardium.[1] The most plausible hypothesis seems to be, that irregular and occasional reflux takes place at the mitral orifice, through disordered action of the muscular apparatus connected with the valve.[2] Just as the external choreal movements sometimes cease for moments or for minutes at a time, so the cardiac murmurs disappear. The main objection to this hypothesis seems to arise from the difficulty of understanding how the heart as a whole can act regularly (as it certainly does in chorea), and yet part of its substance contract irregularly. Still the same contradictory state of things may sometimes be witnessed in the voluntary muscles—portions of which may twitch automatically while the remainder of their substance is quiescent.

The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs, attending dilated hypertrophy of the left ventricle, and disappearing under treatment.[3]

So, too, may be explained a fact, for which it seems otherwise impossible to suggest any plausible theory, that in certain cases of soft, flabby, and possibly fattily atrophous heart, a murmur at the left apex will occasionally be heard, though as a rule completely absent.

Or, again, is it possible that the innervation of the auriculo-ventricular valves may be disordered, so as to impede their closure? They are provided with sympathetic filaments; and (as is admitted to have been demonstrated by Senac and Kurschner) they enjoy a supply, though limited, of muscular fibre: why, then, may they not palpitate as well, for instance, as the aorta?

206. I am aware there are some propositions pretty generally received by physiologists, concerning the part played by the papillary muscles, that appear hardly to warrant such importance being attached to a disordered state of their contractions. Thus, if the heart be removed from the body, an auricle cut away, the artery of the same side tied, and the cavity of the ventricle filled with fluid; and if then a stream of water be directed upon the auriculo-ventricular valve, this rapidly closes. From this experiment of Baumgarten, the inference has been drawn that the auriculo-ventricular valves are closed by the systole of the auricles, prior to the systole of the ventricles, and that the closure is not in anywise influenced by the musculi papillares, but is much facilitated by the specific lightness of the valves themselves. It is further inferred that the papillary columns merely afford a support to the chordæ tendineæ, which in turn prevent the valve from floating too far on

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[1] I may mention here that my experience is far from supporting M. Sée and others as regards any necessarily intimate connection between rheumatism and chorea.

[2] Among numerous illustrative cases may be selected: Robinson, U. C. H., Females, vol. xiv. p. 90; Gosling, U. C. H., Females, vol. xiv. p. 124.

[3] Bunsey, U. C. H., Males, October, 1850.

the auricular side. The multitude of points in which the experiment fails to imitate the natural state of things in the living and contracting heart, gravely invalidates, in my opinion, the conclusion it has been forced to furnish. But, even granting that this inference be sound in *physiology*, the additional assumption of M. Hamernjk, that *morbid* conditions of the muscular structure of the heart can have no effect in preventing closure of the valves, seems a palpable *non sequitur*. What! suppose that (*inter alia similia*) the papillary muscles are shortened, puckered, dwindled in muscular texture, and infiltrated with pseudo-fibrous induration-matter, in a word cirrhosed, will not this state somewhat, at least, interfere with the function of the valves? Why, this would almost amount to affirming that shortening and thickening of the chordæ tendineæ can produce no embarrassment in the play of the valves.

207. I have met with some few instances of inconstant murmur-like quality of the diastolic sound at the base, which appeared to me to be possibly dependent on disordered dynamism of the aortic valves.

208. In certain cases of apparently purely dynamic palpitation, of excessively irregular rhythm and force, murmur-like quality, or actual murmur, may be momentarily heard—now basic, now at the apex, now systolic, now diastolic. These anomalous conditions find their explanation in the foregoing statements. But I must add that I have not actually noticed *well-defined* murmur, constant for any length of time, unless there were, on other grounds, more or less strong reason for the suspicion at least of organic disease.

209. *Of Individual Endocardial Murmurs.*—In endeavoring to ascertain the seat of production of any given endocardial murmur, the essential points in the inquiry are its relationship to the systole or diastole, and the spot of its maximum intensity on the surface of the chest. Subsidiary conditions of great importance are the direction of transmission, duration, clinical progress, quality, and pitch of the murmur; while, in addition to its own characters, the state of the heart's natural sounds, and the presence or absence of certain audible phenomena in the arteries and veins, or both, are valuable subsidiary guides.

210. Each orifice of the heart may be the seat of two murmurs, constrictive and regurgitant—with, or against, the current: the total number of cardiac murmurs connected with the orifices, therefore, reaches eight. The essential characters of these may be briefly set down as follows:—

211. (a.) *Systolic Murmur of the left apex.*—A *systolic* murmur of maximum force at, and immediately above, or to the outside of *the left apex*—but faintly audible, or wholly inaudible, at the right apex (say, the ensiform cartilage), the mid-sternal base,[1] and the

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[1] If the stethoscope be carried gradually upwards and inwards from the maximum point towards the third cartilage, it will in many, but not all, instances be

pulmonary and aortic cartilages—more or less clearly audible about and within the inferior angle of the scapula, and beside the dorsal vertebræ from the sixth to the ninth,[1] audible or not round the lateral base of the chest from the cardiac to the scapular region, is essentially characteristic of *regurgitation through the mitral orifice* at the moment of the ventricular systole. This regurgitation may be the result of inefficiency of the valve, produced by chronic changes of structure (its common cause)—or by enlargement of the orifice without coeval growth of the valve (a *very* rare cause)—or occur from non-closure in acute endocarditis, in consequence of roughness of the edges of the valves and shortening of the chordæ tendineæ—or, probably, as already seen [205], from disordered action of the columnæ carneæ. But it is not absolutely pathognomonic of such regurgitation; for, in some very rare cases, true sacculated aneurism of the heart—fibrinous coagula amid the columnæ carneæ, near the valvular portion of the ventricle—and vegetations on the ventricular surface of the valve, have produced a murmur of this rhythm and site. The dynamic or organic nature of the murmur cannot be determined from its audibleness, or the reverse, round the lateral base of the chest.

Of very variable quality, this murmur is rarely of high pitch, generally oscillating between whispered *who* and *rr;* but I have known it sharply whistling, as of the wind through a key-hole. Once established, it is, as a rule, permanent; but when, probably, dynamic, as in chorea, it may momentarily, as well as permanently, disappear. If the cause of non-occlusion act intermittently, the murmur will be present at some moments, absent at others. This I once observed in an adult male, in whom a body about the size of a large pea was suspended by a thread-like peduncle from the larger division of the valve, in such manner that it might or might not, according to accidental circumstances, have fallen within the orifice, and impeded its closure.

A mitral regurgitant murmur may completely or partially cover the first sound at the left apex. This first sound may have its natural characters in perfection at the base and at the tricuspid apex; but when intense the murmur may partially obscure the sound in both these places by conduction.

In the healthy state, the second sound is more strongly accentuated in the aorta than in the pulmonary artery. The reverse,

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found that at a certain line, defined with singular sharpness, the characters of the murmur completely change: in intensity, it falls to a third; in roughness, loses greatly; in pitch, commonly falls.

[1] I have occasionally known a murmur of indubitable mitral mechanism better audible at the upper than the lower part of the left vertebral groove, and consequently in so far simulating an aortic murmur. This must depend on some unusual condition of the lung and other intervening conducting material. (Dunn, U. C. H., Females, vol. xvi. p. 296.) The murmur may even be *inaudible* at the inferior thoracic part of the groove, while distinct superiorly—*e. g.* Mahon, U. C. H., Females, vol. xv. p. 134. For the topography of the valve posteriorly, vide 34.

according to Skoda, holds in cases, among others, of mitral regurgitation; and the peculiarity is explicable thus: With every systole some blood is forced into the left auricle; that auricle, the pulmonary veins, and pulmonary artery quickly become overstretched, and the right heart requires greater effort to force the blood into the over-filled vessels; the pulmonary artery consequently presses with increased force on its column of blood, and hence intensifies its own portion of the second sound. And, as a corollary, he holds that the absence or presence of this reinforcement will distinguish systolic murmur at the left apex, caused by regurgitation, from that caused by friction of the blood against roughnesses in the ventricle. I do not believe, although it certainly does exist in some cases, that any such implicit confidence can be placed in this sign. I have known it wanting in cases of mitral regurgitation, even when there was no obvious tricuspid regurgitation to afford a plausible explanation, through diminished current, of its deficiency. It seems to me sometimes, too, only a pseudo-accentuation of the pulmonary second sound, from real weakening of the aortic second sound through the lessened current and diminished calibre of that vessel, that follow on long-continued mitral regurgitation. Besides, I have not found thickening or enlargement of the pulmonary artery in such connection with mitral regurgitation, as ought to obtain, were the theory described wholly accurate—the more so, as thickening and enlargement of the left auricle really do exist in a fair proportion of these cases.

Mitral regurgitant murmur may be highly marked with strong, deficient with feeble, systoles.[1]

A murmur, of the character now described, is the most common of the organic class; in rare instances it may be of dynamic mechanism [205], in yet rarer depend on coagulation within the ventricle, but never, so far as I know, arise from altered composition of the blood, anæmic or other. It is always connected either with the mitral valve or orifice, or with the adjoining portion of the ventricle.

212. (β.) *Systolic Murmur at the right apex.—A systolic murmur* of maximum force *immediately above, or at the ensiform cartilage,* inaudible, or nearly so, at the left apex, and, according to my experience, imperceptible in the left vertebral groove opposite the lower angle of the scapula, originates in the *right ventricle.* In the great majority of cases this murmur arises from *tricuspid regurgitation.* It may also by possibility depend, at least theoretically, on sharp collision of blood among the thickened and roughened chordæ tendineæ.[2] In the former case, distension and pulsation of the auricle, vena cava, innominate and jugular veins is habitually present, that

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[1] Allen, U. C. H., Females, vol. xvi. p. 167.

[2] I have never heard a murmur, during life, indisputably proved after death to have been thus generated. Nor, although Skoda and others admit without any apparent hesitation this form of mechanism (Skoda, Markham's Translation, p. 236), I know of no printed demonstration of the fact.

of the latter *visible* but not *palpable;* but the absence of such pulsation does not prove that the murmur under consideration is independent of regurgitation.[1]

This tricuspid murmur is generally soft, and of low pitch, rarely masks the systolic sound completely, is of rarer occurrence than tricuspid regurgitation itself, and is probably not always detected when it exists. It is, absolutely speaking, rare, because regurgitation often occurs from insufficiency, without roughness or other morbid change, of the valves, and because the back current is often not forcible enough for the production of a murmur. On the other hand, it escapes detection, because it is often covered by a powerful mitral murmur, and in some cases impaired in distinctness by deep-seated venous hum. In rare instances, where a mitral and tricuspid murmur coexist, a spot may be found midway between their points of maximum force, where there is comparatively little murmur.

In certain cases of mitral regurgitation, the attendant murmur may be so deadened at the left apex by emphysematous lung, as to be better audible at the right apex, and so simulate a tricuspid murmur.[2]

213. (γ.) *Systolic basic Murmur transmitted to the right and upwards.*—A *systolic murmur*, of maximum force, *at mid-sternum, opposite the third interspace* (or, it may be, the upper part of the fourth rib), abruptly losing force between this point and the left apex, where it may be almost inaudible, faintly perceptible at the second left cartilage, clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposite the second, third, and fourth vertebræ, thence rapidly losing strength downwards, and disappearing about the sixth, originates at the *aortic orifice.*

In some instances, in consequence of the relationship of the vessel to the surface and the nature of the intervening material, this murmur is better conducted to the *left* than to the *right* of the sternum in an upward direction [19]; and, per contra, it may be better audible down the sternum than in the direction of the left ventricle. Again, it may be more distinct in the upper part of the back, to the right than the left of the spinal column—the lungs, mediastina, and arch of the aorta being, as far as evidence goes, healthy.

This murmur is commonly of high pitch, loud, prolonged, and harsh; it may, however, be low-toned, and of peculiar twanging, almost musical quality.[3] If the ventricle behind be dilated and hypertrophous, this state of things increases its intensity, and may give it a drawling, prolonged character, if the sides of the orifice be much contracted.

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[1] Hishin, U. C. H., Females, vol. xi. p. 286; well-marked tricuspid regurgitant murmur, and no actual jugular pulsation.

[2] Hutchinson, U. C. H., Males, vol. vii. p. 335.

[3] Gall, U. C. H., Males, vol. xvii. p. 272.

Although audible at the aortic cartilage, it is distinctly fainter there than at the base; if as marked, and *à fortiori* if more marked there than opposite the valves themselves, disease of the arch of the aorta itself adds to the murmur.[1] The same inference is justified by any notable difference in pitch in the two spots.

A murmur of the attributes, now enumerated, habitually signifies obstruction, smoothly constrictive or rough, of the aortic orifice; in some rare instances it has been traced to fibrinous coagula impeding the onward current. But it must not be forgotten that a systolic basic murmur may be the result of mere anæmia [199]; and that murmurs very closely simulating it may occur in cyanosis and in certain sacculated aneurisms of the heart and small pouches of the aorta, about the sinuses of Valsalva, or elsewhere just above its orifice: the diagnosis and mechanism of these murmurs are considered with the diseases to which they appertain.

214. (δ.) *Systolic basic Murmur transmitted to the left and upwards.*—A *systolic murmur* of maximum force, *at the sternal edge of the third left cartilage*, or a little lower down, loud at the pulmonary cartilage, notably less audible at the aortic cartilage, perceptible (more or less markedly according to the loudness at its point of maximum force) at the upper part of the front chest, but faintly transmitted towards the apex, and almost imperceptible in the back, indicates *obstruction at the orifice of the pulmonary artery*, simple roughness in its valves, or, as noticed by Dr. Elliotson, pressure on the vessel by adventitious masses in the pericardium. All these causes combined, however, are so unusual, that few persons have actually met with such a murmur: some simulating murmurs will be described hereafter. I have only observed one of the kind; and as, in this instance, there was no *post-mortem* examination, its site cannot be held to have been certain, seeing established experience of its characters is so limited.[2]

215. (ε.) *Diastolic Murmur at the left apex.*—A *diastolic murmur*, of maximum force, *immediately above and about the left apex*, and conducted in the same directions, though less extensively, as systolic murmur of the same seat, indicates *obstructive narrowing of the mitral orifice*, or simple roughness of the auricular surface of the mitral valve, or both states combined. Skoda affirms that murmurs of the two sources may be distinguished by the condition of the second sound in the pulmonary artery: it will be loud to excess in the case of narrowing; unaffected in that of simple roughness, unless other causes of reinforcement be present. The distinction

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[1] There is one kind of exception to this statement: if the apex of the right lung be solid, the murmur may be more marked at the aortic cartilage than at the base, though the aorta is healthy. I have never known more than slight excess under these circumstances [157].

[2] A very conclusive instance has lately been recorded by Dr. J. W. Begbie (Beale's Archives, No. v., 1860).

will hence, even if well-founded, be clinically useful only in cases where the absence of mitral regurgitation is certain.

I have never heard this murmur of great intensity, nor high pitch; it is, however, sometimes prolonged. It is rarely loud enough to cover the second sound completely, even at the left apex.

This murmur is commonly spoken of as diastolic in rhythm; but in point of fact it is rather post-diastolic or præ-systolic, than precisely coincident with the diastole.

This murmur is not very unfrequently wanting, where constriction is found after death. Sometimes the deficiency may be fairly referred to the weakness of the auricular systole and smoothness of the constricted orifice;[1] where the constriction is slight, the friction will also be but slight. When deficient, as it has positively been, in cases of marked contraction, Hope thought the deficiency depended on the very fact of the extreme smallness—an explanation not over plausible.

I have known this murmur come and go from day to day in a case where the mitral orifice was very greatly contracted and rigid:[2] probably from varying force of the heart's action.

216. (ζ.) *Diastolic Murmur at the right apex.*—A *diastolic murmur*, of maximum force, at *the ensiform cartilage*, most faintly audible at the left apex, and inaudible at the base, would probably indicate *tricuspid narrowing*, were there a hypertrophous auricle behind that orifice, to give force to the current. Experience concerning this murmur is on a very limited scale: the little I know of it, by actual observation, will be found set forth in the Description of Diseases of the Orifices in Part II.

217. (η.) *Diastolic basic Murmur conducted to the right upwards and directly downwards.*—A *diastolic murmur*, of maximum force *at mid-sternum, opposite the third interspace or fourth cartilage*, conducted (with some exceptions, to be mentioned presently) on the same principle as the systolic murmur of the same site, indicates *regurgitation through the aortic orifice.*

This murmur may be heard with almost as much intensity about the ensiform cartilage as opposite the third interspace—in this point of view differing materially from constrictive aortic murmur: the more distant conveyance downwards in the former case probably depends on the downward direction of the current producing the murmur. So marked is the fact, that unless with care the murmur of aortic regurgitation might be mistaken for one of tricuspid constriction. I have even known the murmur louder at the upper edge of the xyphoid cartilage than at the base of the heart.[1] It is remarkable that the conduction is more perfect towards the right than the left apex—consequently not, as might have been supposed, in the direction of the communicating ventricle: concerning this point I find my experience at variance with the statement of Hope. The murmur may, though well marked in front, be quite inaudible in the vertebral groove. The murmur is in rare cases louder to the left than the right of the sternum on the level of the second cartilages—a special effect of conduction [19].

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[1] In the case of Denham (U. C. H., Males, vol. vi. p. 77), the long tongue of the mitral valve was much thickened; at its union with the other tongue it was puckered; and a mass of ossiform substance, as large as a very small marble, smooth on the auricular, rough on the ventricular surface, had formed at the union of the two tongues. Here there was no mitral constrictive murmur.

[2] Kernis, U. C. H, Females, vol. ii. p. 240.

The second sound of the heart may be covered completely at the maximum point of the murmur; or it may be heard at the beginning of, during, or at the close of this. In the first case, the valves are utterly incompetent: in the varieties of the second case, one division of the valve may flap naturally, or all three imperfectly, and so produce an imperfect second sound; or the second sound heard may be wholly that of the pulmonary valves.

The most common of these compound conditions of diastolic murmur and sound at the base, is that of murmur abruptly brought to a close by sound; a state pretty accurately rendered by the whispered symbols *phwi...tt* or *phwe...tt*—the *tt* being sharply accentuated. Skoda thinks, under these circumstances, the murmur is due to friction of the recoiling blood during the arterial systole against the roughened coats of the aorta, and the sound to the natural closure of the valves without regurgitation. This explanation may hold in some instances; I am certain it will not apply to all. For I have observed this state of basic diastolic sound, where the existence of marked visibleness of all the superficial pulses left no doubt in my mind of the existence of aortic regurgitation—the patients being besides young and free from notable hypertrophy of the left ventricle. In such cases the explanation may then be that partial aortic reflux takes place, the valves being partially, not wholly, disabled from closing. Or it may be that no diastolic sound is produced at the aortic orifice at all, only feeble murmur—while the sound heard is that of the natural flap of the pulmonary valves, a little sequential in time: but the impression on the ear is so strong of the two divisions of the *phwe...tt* sound being produced at the same spot, and being distinctly connected with each other, that it is difficult to accept this explanation.[2]

To return to ordinary diastolic murmur: unless the murmur be of very great intensity, the second heart-sound may be well heard at the left apex: it may even be strongly accentuated there[3]—a fact which has already [149] been made the subject of comment. The state of the second heart-sound at and about the second left, or pulmonary, cartilage, varies: it may be well or feebly audible, or wholly imperceptible—a variation to be explained by the intensity and mode of conduction of the murmur in different cases.

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[1] Flood, U. C. H., Males, vol. vii. pp. 265, 266.

[2] In one case of the kind (Monk, U. C. H., Males, vol. xii. pp. 72, 86), there was well marked reticulation of the aortic valves. It is, perhaps, conceivable that this condition might allow of reflux at the *commencement* of the time of closure of the valve.

[3] Flood, U. C. H., Males, vol. vii. p. 265.

Aortic regurgitant murmur is usually of blowing quality, sometimes almost hissing, generally of low pitch, rarely rough, weak as a rule (though I have known it extremely loud, and it is said to have been heard at a little distance from the surface), occasionally of twanging, almost musical, character.

This murmur occasionally seems to pass from the direction of the ausculting ear: this will be the case whenever the stethoscope happens to be placed higher than the heart's base. As a rule it is well prolonged, habitually filling the post-diastolic silence.

The causes of this regurgitation are the ordinary morbid changes entailing incapacity of valves; I have known sudden rupture of one of the sigmoid valves produce it.[1] Reticulation of the valves carried to extremes, a conceivable source of murmur, has never fallen under my notice as its cause (vide VALVULAR ATROPHY); but perforative destruction on a large scale will surely generate it. Mere insufficiency of valves, healthy in themselves, but too small to fill the morbidly widened mouth of the aorta, has in rare instances produced this murmur. In exceptional cases it is developed during the *acute* period of endocarditis. It remains for future inquiries to determine, also, whether temporary reflux with its murmur may not be caused by dynamic imperfection without structural change in the valves [207].

218. (θ.) *Diastolic basic Murmur conducted to the left upwards.*—From experiments on animals, it has long been known that a *diastolic* soft prolonged murmur, audible to the left of the sternum above the heart, and conducted down the right ventricle, may be produced by artificially rendering the *pulmonary valves insufficient.* But this regurgitant murmur is of excessive rarity in man; and the few recorded examples have, as a rule, undergone but slight clinical examination. As already mentioned [217], we occasionally find an aortic regurgitant murmur more distinct at the second left than the second right cartilage—an obvious source of fallacy. And, in point of fact, the diagnosis of pulmonary reflux murmur is rather to be made with surety through the absence or presence of certain concomitant conditions, than by means of its own intrinsic properties.

219. In respect of relative frequency, I should be disposed to place intra-cardiac murmurs of *organic* origin in the following order, commencing with the most common: mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.

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[1] Gordelier, Consumption Hospital, Males, vol. i.

These murmurs may be variously associated; the following combinations are arranged in the order of their relative frequency: aortic constrictive and mitral regurgitant; aortic constrictive and regurgitant; mitral regurgitant and aortic regurgitant; mitral regurgitant, aortic constrictive and regurgitant; mitral regurgitant and obstructive; mitral regurgitant and tricuspid regurgitant; mitral regurgitant and constrictive, aortic constrictive and tricuspid regurgitant; tricuspid constrictive and mitral constrictive.[1]

220. The existence of no one organic murmur involves, as matter of necessity, the presence of another; a direct murmur may exist at any valve, and an indirect be absent, and *vice versâ*.

221. When two murmurs co-exist at the same orifice, they are, as a rule, readily distinguishable by their rhythm, their quality, their pitch, and by their appearing to pass towards, or from, the ear. I have great difficulty in believing, with Skoda, that mitral systolic and diastolic murmurs, forming one quasi-continuous noise, shall be in nowise distinguishable from a single murmur, until slackened action of the heart distinctly separates them into two.

B.—PERICARDIAL MURMURS.

222. Pericardial murmurs are divisible, in respect of quality and of the mechanism of their production, into four chief species: (*a*) Friction or attrition murmurs; (*b*) continuous murmurs; (*c*) clicking murmurs; (*d*) murmurs produced by bending of layers of exudation matter. Roughness of surface is the essential statical condition of the three first—movement the essential dynamic element of all four.

223. (*a*.) Attrition murmurs, all of them, more or less, distinctly suggestive of rubbing of surfaces of variable character against each other, occur in a greater number of varieties even than pleural friction-sounds. They resemble, for instance, *grazing*, *coarsely rubbing*, *grating*, *scratching*, *creaking*, *squeaking*, *and prolonged whistling* noises. Traceable as all these varieties commonly are to collision of surfaces roughened with lymph, any interest attached to their distinction must turn mainly on their being severally connected with some particular state of that lymph. Now, experience does not show any such necessary connection; though it be true, for the most part, that at the very earliest period the murmur is of grazing quality, like the sound produced by rubbing pieces of silk together, and that as the exudation hardens, and gathers into irregularly peaked elevations, the quality becomes coarsely rubbing, grating, creaking, this sequence of changes is subject to constant exceptions. Creaking friction-sound, so loud as to be audible *three inches from the auricular end of the stethoscope applied to the surface in the ordinary way*, may depend, I know from actual observation, on tough exuda-

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[1] The anatomical conditions of this latter singular combination are exemplified by Carswell's Drawings, U. C. Museum, Portfolio A, No. 255.

tion-matter with fine rough elevations; and I have noticed distinct, though slight, creaking quality, when the exudation-matter was found to be of almost creamy softness. Dr. Taylor,[1] too, has known signs of moderate liquid effusion coexist with friction-sound of this quality—a fact showing that peculiarly rough attrition is not required for its production.

224. (*b.*) When exudation-matter and fluid coexist, the former imprisoning the latter in its meshes, the heart's action produces a peculiar continuous rumbling, or squashy churning sound, just such as we find occasionally occurs in the pleura. This is rare; but, once heard, can never be forgotten.

225. (*c.*) Occasionally sounds are heard of peculiar clicking character, one or two, with each beat of the heart, which are only distinguishable, at the time, from modifications of the valvular sounds, by their non-synchronism with these, and by the extreme irregularity of their occurrence. I have satisfactorily traced these clicks to the pericardium, and further, in all probability, to the separation, without attrition, of surfaces glued together with exudation matter.[2]

226. (*d.*) It has appeared to me that sound is sometimes generated in layers of firm exudation-matter, though so perfectly agglutinated together that attrition or separation of the apposed surfaces is physically impossible. The quality under such circumstances is probably variable: in the only positive instance of the kind which I have observed, it was faintly creaking. The bending and crumpling of tough exudation-matter may conceivably generate such sounds.

227. Pericardial murmur of one or other variety may be heard over all parts of the cardiac region from the roots of the large vessels to the apex. But I have never detected the churning variety except above the apex, nor the clicking variety except in the site of the large vessels. The finest shades of grazing sound are most common behind the sternum. Different qualities of murmur may be heard at one and the same time in some cases over different parts of the heart; but this is not common. As a rule, friction-sound is most clearly and frequently detected below the third interspace, probably simply because, below that part, there is little lung intervening between the pericardium and surface. Limited in some cases to a mere point of surface, it may reach from the clavicles to the epigastrium and from nipple to nipple.[3] In rare instances, friction-sound in the pericardium is audible in

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[1] Brit. and For. Med. Rev., vol. xxiv.

[2] Barr, U. C. H., Males, vol. vii. pp. 328–9. Here there was clicking pericarditic murmur at the second left cartilage and interspace; although the apex was raised, and the præcordial region bulged by fluid. Also Richmond, U. C. H., Females, vol. xv. p. 5.

[3] Case of Mr. S., seen with Dr. J. H. Davis. The friction-murmur was in this case of the most unmistakable kind—loud, grating, rubbing, jerking. This case alone would suffice to prove that friction-sound is not of necessity limited to the præcordial region.

the back, between the scapulæ and the spine—I mean in cases where mere intensity of sound cannot be held to explain the fact. It has been found single in the back, too, while double in front. It seems sufficiently likely that in some, at least, of the cases where it is alleged to have been confined altogether to the back, the friction heard may have been produced in an inflamed pleura by the cardiac impulse. Pericardial friction is usually abruptly limited.

228. Pericardial murmurs vary exceedingly in intensity; so delicate in some instances, especially when of grazing or clicking quality, that the closest attention is required for their detection—in other cases they may be heard in the posterior and lateral regions of the chest, and even, as already mentioned, at a considerable distance from the surface of the chest in the præcordial region [223]. On the whole, their intensity is greater than that of pleural friction-sounds—a fact accounted for by the comparative abruptness and energy of the motion inducing them.

229. When the entire cardiac surface is the seat of friction-murmurs, the maximum amount of sound exists, according to some writers, about the nipple; to others, behind the sternum. I am satisfied no rule of the kind can be established: I have known the point of maximum intensity change from the fourth interspace to the fifth, thence to the ensiform cartilage, and thence to the nipple, within twenty-four hours.[1] Neither can any particular part of the cardiac surface be fixed on as the absolute seat of loudest friction-sound; it is sometimes, though rarely, excessively loud even about the base.

230. Pericardial friction may accompany both the systole and diastole, or either singly. Its coexistence with the systole alone is not rare; with the diastole alone, infrequent. When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule; the friction-murmur may be very distinct during the post-diastolic silence; and occasionally falls between the first and second heart-sounds. In point of fact, want of distinct synchronism of the murmur with either of the two sounds is a very habitual and distinctive attribute. Friction coexistent with the systole is generally, but by no means constantly, sharper and louder than with the diastole.

231. Pericardial murmurs are so deficient in tone, that their pitch can scarcely be estimated even rudely, although they embrace in their different varieties a considerable compass of sounds. And, indeed, no practical hint, that I know of, is derived from their pitch; except that, generally speaking, the higher this is, the drier and rougher the material of attrition. The pitch of a pericardial

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[1] Campion, U. C. H., Females, vol. vi. p. 260. Here, at one time, it was loudest at the base.

murmur may sometimes be raised by pressure with the solid stethoscope, and also by full inspiration.[1]

232. Pericardial murmurs are, as a rule, distinguished by their superficial character: they appear to be produced immediately underneath the integuments.[2] But if the physical cause of the murmur be placed beyond the limits of the triangular portion of the heart, uncovered by lung, they lose this character, and seem of more or less deep origin. This is sometimes particularly observable about the large vessels, before any effusion has occurred to push the edges of the lungs aside; their apparent depth then contributes to assimilate them in certain varieties to valvular murmurs.[3]

233. If the præcordial interspaces be hollow from emaciation, the heart's action may produce a rustling sound by causing friction of the skin against the stethoscope. In a case of agglutinated pericardium, now under observation, this sound, well audible during expiration, when the surface of the intercostal plane rises against the end of the stethoscope, placed on the two contiguous ribs, ceases altogether while that plane sinks inwards during the succeeding inspiration.[4]

234. Various circumstances modify the intensity and superficial extent of these murmurs—the physical condition of the pericardium itself remaining the same. The most important of these is the energy of the heart's action: the greater this, the louder the friction-sound. The weakness immediately ensuing on blood-letting, approaching syncope, the action of digitalis and aconite, all lower its intensity: if successive systoles be of very unequal force, friction, absent with one, is present with another, impulse. An hypertrophous and dilated heart gives, *cæteris paribus*, the maximum quantum of attrition-murmur. Attrition-murmurs are more marked in expiration than in inspiration; and when the trunk is bent forwards, than in the recumbent position. If there be fluid present, the friction-murmur may be less marked at the lower sternal region in the sitting than the recumbent position, doubtless from gravitation of the fluid.[5]

235. Pericardial friction may appear very rapidly after the cause of inflammation has acted. Thus, in the remarkable case at University College Hospital where fatal perforation of the œsophagus and pericardium was produced in the attempt to swallow a sword, friction was detected by my then Clinical Assistant, Mr. Tidmas, thirty minutes after the accident.

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[1] Bingley, U. C. H., Males, vol. vii. p. 114.

[2] Calkin, U. C. H., Females, vol. x. p. 338, Nov. 1854.

[3] Skoda attempts to show that the clinical distinction of sounds, produced deeply and superficially, is a delusion. If a stethoscope be placed on the chest of ead body, the sounds produced by tapping different parts of the inner surface he parietes, near and distant, will, as far as I have been able to ascertain, r very sensibly: exceptions occur, it is true; but these do not destroy the rule.

Aubrey, U. C. H., Males, vol. xvii. p. 268, Nov. 1860.

son, U. C. H., Females, vol. xv. p. 176.

236. The duration of friction-murmur varies very greatly. I have known it to appear and disappear finally (and inasmuch as the disappearance was final, it could not have depended on the occurrence of liquid effusion), within six hours—of the grazing variety, it is true, but still of unmistakable characters. In illustration of the other extreme, I may refer to a case in which it continued audible for upwards of three months, especially at the lower part of the sternum.[1]

The total disappearance of pericardial murmur may, if it be slight in amount, almost immediately follow general or local bleeding; this disappearance may be only transitory, however, from weak action of the heart: or a pericardial murmur may abruptly disappear from rapid agglutination of the entire of the affected surfaces. I have known it impossible to find a vestige of friction in a case where, six hours before, the *entire* cardiac region was the seat of intense rubbing sound. But, generally speaking, the progress of agglutination is materially slower than this, and for several days one or more limited spots may be found, where the collision continues soniferous (sometimes in the churning variety), after the adhering process has commenced. The sudden cessation of friction-sound, through absorption of exudation-matter, is less easily conceived; and where such cessation occurs, independently either of agglutination, or of rapid pouring out of effusion, the inflammation had probably produced very little indeed of that matter.

237. In the majority of cases where friction abruptly disappears, the change depends on fluid effusion, separating the pericardial surfaces from each other: it is consequently oftener an evil, than a good, sign. The amount of liquid required to annul friction-sound varies with collateral circumstances, such as the size of the heart, conformation of the chest, absence or presence of adjoining pleural adhesions, and the accident of partial soft adhesions having, or not having, occurred, before the fluid begins to accumulate. A case of Dr. Taylor's (loc. cit.) shows that friction may continue when eight ounces of fluid, or thereabouts, have accumulated: but generally less removes it. In a remarkable instance under my own observation friction-murmur was well heard a few hours before the death of an individual whose pericardial sac contained sixty fluid-ounces of liquid effusion.[2] As fluid accumulates, changes may sometimes be traced in the characters of the friction-sound—increasing weakness and softness being their predominant character.

Disappearing with the occurrence of effusion, friction-sound pretty frequently returns when the fluid is absorbed: this returning or redux friction generally, but not always, appears first about the great vessels and base. Pleuritic friction is very commonly caught at the redux period: pericardial, certainly, more generally at the outset of the inflammation. The second disappearance of friction may be abrupt, slow and gradual, irregular with recurrences, or rapid over the heart generally; while in a single spot or two, most commonly either at the apex or about the great vessels, some form of the murmur remains for a time.

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[1] Kennedy, U. C. H., Males, vol. i. p. 67. First heard on the 27th Oct., it was last heard on the 5th Feb. following, long after the man's discharge from the hospital, and apparent restoration to health. How much longer it may have continued I know not, as the man was not afterwards examined, though often seen again on duty in the streets as a policeman—a fact showing the completeness of his recovery.

[2] Bartlett, U. C. H, Males, vol. iv. p. 292.

288. Friction in the pericardium, the cause of which lies within itself, signifies inflammation of the membrane. It is exceedingly probable that mere dryness of the surfaces will suffice to produce the grazing variety. I know from observation that fine vascularity of a very small surface, without a particle of lymph, may produce faint rubbing noise,[1] but exudation matter is its common material element. In the great majority of cases, exudation matter forms on both serous surfaces. Dr. Taylor relates a case where, the cardiac surface alone being affected, ordinary friction was almost completely absent: still, however, it appears to have existed, single and systolic, to a slight amount about the base. If exudation exist on the posterior aspect of the sac only, friction will commonly be inaudible. And, in those rare cases where the product of inflammation in the sac is essentially purulent or strumous, the collision of the surfaces may be totally noiseless.

A former attack of pericarditis will or will not prevent the development of friction-sound with a new attack, according to the state in which it has left the serous membrane and cavity of the sac. If it have left a state of perfect agglutination behind it, then, unquestionably, friction is impossible: if of loose adhesions, new lymph may be thrown out between these, and friction will be developed.[2] But, probably, old pericarditis will always have a tendency to limit the extent and regulate the site of new friction-sound.

Calcification of exudation matter within the pericardium will certainly,[3] and tuberculous and carcinomatous disease may be conceived to, produce permanent friction-sound; but I have not observed this in the two latter diseases.[4] Whether fibrinous, cancerous, or other matter within, and in the walls of, the heart, may, by simply elevating the cardiac surface, produce friction-sound, independently of pericardial irritation, I do not know from experience.

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[1] F. Parker, U. C. H., Males, vol. iv. p. 177, Dec. 1848. A rub may attend the impulse at the apex with knocking sound, independently of any pericardial irritation, as far as can be even suspected; e. g., cases of Sus. Roberts and Bonsey, U. C. H., Oct. 1850.

[2] Craddock, U. C. H., Males, vol. vii. p. 336, admitted for his eighth attack of rheumatic fever: for five of these he had been treated in the hospital, and had pericardial friction on every fresh seizure.

[3] Jones, U. C. H., Males, vol. xvii. p. 315.

[4] On the contrary, indeed, I have known very positive elevation of the pericardium by carcinomatous nodules unattended with the least friction-sound; but the serous surfaces were smooth. Case of R. Smith, Multiple Subcutaneous Cancers, "Med. Times," August, 1852.

239. *Friction-sound of cardiac rhythm produced in the inflamed pleura.*—The distinction of pericardial from neighboring pleural friction, turns mainly upon its difference of rhythm. But sometimes cardiac action produces friction in an inflamed pleura adjoining, the pericardium being unaffected. And I am disposed to believe, but so far on the evidence of only one case, that if the pleural lymph have by its contraction puckered both the pleura, on which it lies, and an adjoining part of the pericardium adherent to itself, the occurrence of cardiac friction-sound of this anomalous mechanism will be materially facilitated.[1] The distinction of this kind of friction, pleural in site, and cardiac in rhythm, is not always easy: though commonly limited to the confines of the cardiac region in front, it may be audible in the back on the left side;[2] its intensity is generally increased by the act of breathing—the pleural surfaces being then in motion of one rhythm, are more easily thrown into motion of another, than if at rest; it seems sometimes limited to the time of expiration.[2] The following circumstances argue in favor of friction of cardiac rhythm being of pleural, and not pericardial, origin: the limitation of the sound to either edge, generally the left, of the cardiac region; fixity in one or more particular spots; cessation complete, or, what is more common, occasional with certain beats of the heart, when the breath is held; and marked unsteadiness in the intensity and quality of the friction-sound. Local dry pleurisy, close to both sides of the heart, and productive of friction both of pleural and of cardiac rhythm, is sometimes followed by dry pericarditis: the presence of the latter inflammation can scarcely be affirmed, until effusion takes place; for the distinction of the true pericarditic friction-sounds is then most difficult.[3]

240. *Distinction of pericardial from endocardial murmurs.*—Pericardial murmur is, in the great majority of cases, easily distinguished from that of endocardial origin by the following characters and circumstances: its rubbing quality; its superficial character [232]; its abrupt limitation and non-transmission in the course of endocardial murmur; its changeableness in precise seat and intensity from hour to hour; the increase it undergoes in sharpness and extent when the patient bends forward; its want of perfect synchronism with, or fixed relationship to, the heart's sounds; and its being sometimes accompanied by friction-fremitus, which can scarcely be confounded with valvular thrill. Pericardial murmurs

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[1] Shears, U. C. H., Males, vol. xv. p. 87.

[2] Wilkinson, U. C. H., Males, vol. ix. p. 284. But true pericardial friction, as already mentioned [234], is commonly most marked in expiration, *e. g.*, W. Price, U. C. H., Males, vol. vii. p. 214.

[3] Two cases of this kind (Hayes and F. Smith) once fell under my notice at the same time—U. C. H., Ward 4, October, 1850. Vide Clin. Lect., "Lancet," loc. cit. p. 389, 1849.

are commonly rougher than *acute* endocardial murmurs. Hope, indeed, held that a diastolic *rough* sound is of necessity pericardial, endocardial diastolic murmurs never possessing this character; but in this he was absolutely wrong: an aortic diastolic murmur may be very rough. On the other hand, cases occur where it is next to, or absolutely, impossible to decide on the peri- or endocardial origin of a murmur. This difficulty is oftenest felt about the base and great vessels, and when the sound is of clicking character. Sounds really produced at the valves of the aorta may not be transmitted along this vessel—as a consequence simply of their feebleness. Again, if both divisions, systolic and diastolic, of a double murmur be loudest at the same spot, that murmur is probably pericardial; for, generally at least, the divisions of a double valvular murmur attain their maximum intensity at points slightly different. It has been said, if the heart's sounds be well distinguished and in a *perfectly unchanged condition* through a synchronous murmur, the fact argues for pericardial mechanism: I believe that there is some truth in this proposition, fairly interpreted; but degree of perfection of audibleness of a sound through a murmur is a matter so delicate, that it cannot be clinically trusted to. Full inspiration will sometimes notably raise the pitch of a pericardial murmur [231], but to a less degree it may, by possibility, similarly affect an endocardial.

And, in truth, it must be confessed that pre-cardiac murmur sometimes presents characters so wanting in intrinsic distinctness, that the diagnosis of pericardial or endocardial origin is almost wholly to be made from the course and mode of progress of the murmur and upon the attendant conditions. According to some observers the diagnosis may be established in difficult cases by the different effects on the murmur produced by stethoscopic pressure —increase of intensity being the attribute of the pericardial class. The distinction, I am satisfied, cannot thus be made; and, as the present seems an appropriate place, the general subject of the influences of stethoscopic pressure on the characters of murmurs may here be briefly considered.

241. *Influence produced on murmurs of the pericardium, endocardium, or orifices of the great vessels by stethoscopic pressure.*—The following propositions embrace the results of my experience on this subject: (1.) Whatever be the effect of pressure in any particular instance, that effect is better marked with the solid than the hollow stethoscope. (2.) In the majority of cases an existing murmur, whether endocardial or pericardial, reaches the ear with greater intensity if the stethoscope be firmly pressed against the chest, than if it be lightly applied. (3.) This increase of intensity is in part the result of the more perfect conduction effected by firm application of the conducting medium. (4.) But in some cases there is actual augmentation of mass of sound. (5.) This augmentation is more frequently observed in the case of pericardial friction than of other murmurs; and in certain instances of superficial grazing friction,

where the collision of the pericardial surfaces is slight and consequently the resulting noise slight, both collision and noise may be greatly increased by pressure. (6.) But *per contra* the very reverse effect will ensue, if it happen that pressure be made in a case where the already existing attrition is precisely of the amount best calculated to produce noise; if the freedom of attrition in such a case be interfered with by approximating the rubbing surfaces too closely, the amount of noise will be lessened.[1] (7.) Pericardial rubbing-sound may in persons with flexible chests be often artificially produced by pressure, though the membrane—free from exudation or morbid vascularity—be in fact perfectly healthy. (8.) The pitch of a pericardial murmur is sometimes distinctly raised by pressure. (9.) The quality of a pericardial murmur may be rendered more grating by the same process. (10.) That a true endocardial murmur may be reinforced by pressure has already been mentioned,[2] but it may occasionally be thus produced *de novo* (when inaudible by the ear applied directly to the surface) by very forcible pressure. (11.) I have not known such production of murmur within the heart synchronous with the diastole: the phenomenon, as far as I know, is always systolic. (12.) The most common seat is the base (therefore at the orifices of the great vessels), or at the second left space and cartilage in the direction of the pulmonary artery. I have observed artificial murmur also in the second right space. (13.) Thinness and flexibility of chest-wall are important conditions of the occurrence in all positions, but especially to the right of the sternum. (14.) The murmur at the left second space is of course excited in the pulmonary artery; that in the second right in the aorta; that at the base, probably, on account of its greater proximity to the surface, in the pulmonary artery. (15.) I believe that I have heard systolic murmur at the left apex, having the attributes of the mitral regurgitant variety produced wholly by pressure. But I have no post-mortem evidence to prove the proposition; and it is difficult enough to understand how the two things should stand to each other in the relationship of cause and effect. Still very certainly a mitral murmur may even in the adult be notably increased by pressure.[3] (16.) In exciting endocardial murmurs by pressure on either side of the sternum, it is important to use a stethoscope of small diameter at its applied end, so that this may be partially placed between the ribs. I have never observed an artificial murmur to the *right* of the sternum except when an instrument of this kind was used.

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[1] Lower, U. C. H., Females, vol. xiv. p. 259. Scratching superficial pericardial friction wholly stopped by pressure.

[2] Lewis, U. C. H., Females, vol. ix. p. 320; Gay, U. C. H., Females, vol. ix. p. 316; in both cases systolic basic. This fact was, indeed, long ago pointed out by Dr. Latham (Clin. Lect., vol. i.), who cautions the observer against being deceived by such artificial murmurs.

[3] Beere, U. C. H., Females, vol. x. p. 90; Craddock, U. C. H., Males, vol. x. p. 83.

(17.) The pitch of an already existing endocardial murmur is commonly raised by pressure. (18.) The pitch of an endocardial murmur, wholly artificial, is commonly rather high; its quality blowing.

242. It follows then with sufficient clearness from these propositions that the mere fact of increase of intensity taking place by pressure will not distinguish with certainty endocardial and pericardial murmurs from each other. But if by pressure the apparent area of generation of a murmur be increased, and above all *its rhythm changed*, if from single it be changed into double,[1] then I am disposed to believe the proof of pericardial origin is conclusive.

243. The influence of *external* pressure in modifying already existent, and in generating new, murmurs, is illustrated by the analogous effects of *internal* pressure of various kinds and mechanisms. The more important of them may be grouped in the following manner:—

A. Pleuritic effusion displacing the heart sidewards and twisting more or less the great vessels at their roots, occasionally gives rise to systolic, and in infinitely rare instances even to diastolic, murmur. Of the latter a very unquestionable example, as I think, has been referred to in another place.[2]

B. Mediastinal tumor pressing on either the pulmonary artery or aorta, and an aneurismal sac of the aorta bearing against the pulmonary artery, may give rise to systolic blowing murmur.

C. Pericardial exudation-matter embracing the great vessels tightly at their roots, and constituting another variety of pressure, may engender systolic murmur in the aorta or pulmonary artery, or both. So, too, accumulation of fluid in the pericardial sac pressing on the great vessels may, according to Martin-Solon, exercise a like effect.

D. Certain enlargements of the abdominal organs, pushing the heart upwards, in some instances appear to engender systolic murmur in the great vessels. But no such result necessarily follows; and in certain reported instances of this alleged mechanism, the question is encumbered with the possible influence of spanæmia.

III.—ANOMALOUS EXTRA-CARDIAC SOUNDS PRODUCED BY THE HEART'S ACTION.

244. The action of the heart sometimes produces rhonchal sounds in the adjacent lung—cavernous, and coarsely or finely bubbling. The rhythm of the rhonchi, and their persistence during suspension of the breath, disclose their mechanism.

245. A double rasping friction-sound, so loud as to be audible at two inches distance from the chest, existed in Dr. Swett's remarkable case of enlarged granular liver, where the pericardium and valves were perfectly natural [87].

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[1] Pearse, U. C. H., Males, vol. xv. p. 60, June, 1858.

[2] Diseases of the Lungs, 3d Am. Edit., p. 212.

IV.—RESPIRATORY SOUNDS AS AUDIBLE IN THE CARDIAC REGION.

246. In the natural state the respiration is clearly audible over the entire cardiac region, though somewhat enfeebled below its centre. Enlargement of the heart widens the superficial extent of this feebleness; pericardial effusion does so also. Now, in proportion to the percussion-dulness produced, liquid accumulation enfeebles the respiration-sounds much more than solid heart-enlargement—to so sensible an amount, indeed, that the excess is clinically useful *as an aid* in distinguishing these two states of disease under difficult circumstances. The difference in solid and fluid conduction explains the clinical fact.

Pericardial adhesions, especially if attended with close union of the pericardium to the ribs, and agglutination of the adjoining pleural surfaces, will maintain the natural amount of audible respiration in the pericardial region, through the course of a subsequent attack of pericarditis with liquid effusion. Under these circumstances, persistence of respiration in front of the heart would, as I have elsewhere suggested, take rank as a sign of pericardial adhesions.[1]

V.—VOCAL RESONANCE IN THE CARDIAC REGION.

247. In the state of health, vocal resonance is weak, it may be actually null, in the præcordial region. In some cases of pericardial effusion, the resonance is not only morbidly loud on the confines of the liquid, but acquires an ægophonic quality. In the only instance in which I have actually observed this, the state of the lung adjoining the distended sac accounted for the increase of resonance—the fluid simply gave this an ægophonic quality and *pro tanto* raised the pitch.[2]

[1] Clin. Lect. "Lancet," loc. cit., p. 144, 1849.

[2] Harley, Consumption Hospital, Males, vol. i. p. 76.

§ II.—GREAT ARTERIES.

SECTION I.—INSPECTION.

248. THE outline and movements of the large arterial trunks are not visible to the eye in the state of perfect health. In very emaciated persons the pulsation of the aorta in the epigastrium may be seen, however; and, if the arch of that vessel lies unusually high, and the individual be thin, slight impulsive motion may be visible above the sternal notch. The carotid pulses can scarcely be seen, if the neck be tolerably well provided with soft parts: the same statement applies to the smaller arteries. No visibly expansile character can be detected in any of these motions.

249. With these exceptions, notable visibleness of arterial pulsation, especially with onward locomotion of the artery, is an unnatural condition, depending on (*a*) general or local excitement of the circulation; (*b*) special disease of the heart; or (*c*) disease of the visible vessel itself.

250. (*a*.) General excitement of the circulation, it is well known, produces visible throbbing action in the innominate artery and carotids, which passes away with that excitement. The vessels, however, simply pulsate, and undergo no onward locomotion. Visible pulsation of the epigastric aorta may be similarly induced. The arteries leading to an inflamed part often beat visibly.

251. (*b*.) Aortic regurgitant disease, as first shown by Dr. Corrigan, renders the pulsation of the superficial vessels visible: the vessel moves forward too, in the direction of its axis—vermicularly, if the outline chance to be tortuous. The radial arteries at the wrist, the temporals, and the posterior tibials behind the malleoli are the usual seats of the appearance; but in highly marked cases the carotid, brachial, axillary, femoral, and external iliac arteries distinctly present it.

To what extent may this sign be trusted to, as evidence of aortic regurgitation? In the first place, no case of that disease, well defined in *all* its other clinical characters, has ever fallen under my notice in which visibleness of the superficial pulses was not more or less obviously present. But I have known murmur, basic and diastolic (and obviously from its concomitants not seated in the pulmonary artery), where the radials were not visibly pulsatile, though the patient was thin.[1] Such cases are very rare; but they certainly do constitute exceptions to the rule. In the second place I have never observed *highly marked and extensive* visible pulsation without aortic regurgitant disease. Hope thought that where there was mitral regurgitation or contraction to any amount, aortic regurgitation failed to render the pulse visible. This seems somewhat theoretical, and is positively opposed to what I have seen.[1] But in the third place, in aged persons of thin frame, whose vessels are calcified and tortuous, a slight degree of the phenomenon may certainly be noticed in the smaller arteries, independently of any affection of the aortic valves; and if the left ventricle be hypertrophous in such persons, the amount of visibleness will be materially greater; but still, as far as I have seen, will fail to affect the larger trunks. I have very rarely[2] known simple or dilated hypertrophy alone produce this sign in persons under the age of forty, whose limbs were even moderately well covered with flesh. If this qualification regarding age be borne in mind, exceptional cases will, I think, be found to be singularly few. In the fourth place I have occasionally seen cases of marked emphysema, unattended with the least clinical evidence of aortic disease, where the majority of the superficial arteries, even the femorals, pulsated visibly.[3] In the fifth place, in a case which presented all the clinical requirements for the diagnosis of coarctation of the arch of the aorta, the diastolic heart-sound being healthy, several of the superficial arteries were visibly locomotive.[4]

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[1] S. Dunn, U. C. H., Females, vol. xvi. p. 296.

252. In all these exceptional cases the symmetry of the phenomenon has appeared to me to be less perfect than when dependent on aortic regurgitant disease.

253. (c.) The impulse of arteries locally diseased is much better studied by the touch than by the sight.

SECTION II.—APPLICATION OF THE HAND.

A.—IN HEALTH.

254. The systole of the ventricles conveys to the entire extent of the arterial system an undulatory and somewhat expansile motion, easily felt in vessels, of a certain size, lying within reach of the fingers, and known as the arterial pulse or diastole. Each arterial diastole is followed by a contraction or systole. In vessels close to the heart the arterial diastole is perfectly synchronous with the ventricular systole, but falls more and more behind it, in point of time, the further the artery from the central organ. With this

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[1] Denham (U. C. H., Males, vol. vi. pp. 69—78) had marked mitral regurgitation and constriction associated with aortic regurgitation, yet his radial, brachial, posterior tibial and femoral pulsations were well visible.

[2] Beere, U. C. H., Females, vol. x. p. 98. Ætatis 40—visible pulses, hypertrophy of left heart, without diastolic basic murmur.

[3] Slater, U. C. H., Males, vol. xii. p. 79; neither aortic regurgitation nor hypertrophous heart, ætatis 44.

[4] Gunnee, U. C. H., Males, vol. xiv. p. 38.

qualification, the synchronism of the pulse and ventricular systole is perfect in health; the altered rhythm of the latter, produced by changes of posture, and all other physiological causes, is impressed on the former. In diseases of the heart, various perversions of this synchronism occur, and have been already described [172]. The systole of the arteries, synchronous with the heart's diastole, is normally pulseless.

255. The characters of the pulse which may have diagnostic signification in cardiac diseases are as follows: the diastole may be quick or slow; short or prolonged; soft or hard; loose or tense; empty or full; small or large; equal or unequal in force, and other characters, in successive beats; and the rhythm may vary in the different forms elsewhere noted [172]. The stroke may be vibratory, jerking, undulatory, bounding, or reduplicate (bis-feriens), the systole being then pulsatile; these terms are in themselves sufficiently explanatory of the states to which they refer.

256. The pulsation of the thoracic aorta can be felt only in one spot in health—above the sternal notch. If the finger be pressed downwards in that position, the patient's head being at the same time bent forwards, slight impulse is, in the majority of cases, detected: where the arch lies high, there will, of course, be very distinct movement—sufficiently marked, indeed, to suggest the notion of possible dilatation of the vessel. True aortic impulse is felt directly in the middle line; impulse inclined to the right side originates in the innominate artery. The pulsations of the abdominal aorta may be felt, if pressure, proportional to the thickness of the parietes and intervening viscera, be made with the hand or stethoscope. The movement is simply one of elevation, without expansile action, of variable force. Except in very thin persons, it is impossible to feel the beat of the vessel laterally.

B.—IN DISEASE.

257. In the various forms of dilatation of the arteries, the dilated portion pulsates with undue force, unless it be more or less completely filled with solidified fibrin. The character of the movement becomes *hammering*—its force sufficient in some cases to shake the entire trunk and limbs. Besides, the diseased vessel becomes, under certain circumstances, the seat of a double impulse with each contraction of the left ventricle; the arterial systole conveys to the hand an appreciable shock. The diastolic pulsation is expansile, as well as heaving, in character; but the distinction of the former quality of impulse is not always practically easy. Now, in the case of the thoracic aorta, there is no artifice by which obscure expansion may be rendered distinct to the touch: in that of the abdominal portion of the vessel, the hands may be sometimes slipped deeply on either side of the vessel, and a double sideward impulsion sometimes detected, which escapes discovery when the examination is made in front only. Still, it must be confessed, the pulsation of

the healthy aorta, when pushed forwards by an enlarged vertebra or tumor, cannot always be distinguished by this plan from that of enlargement of the vessel itself.

258. *Arterial thrill.*—Arterial pulsation is in various forms of disease accompanied with thrill, occasionally more intense than that of valvular origin. Simple peripheric dilatation of a vessel, especially if it be roughly calcified, is a more efficient cause, than sacculating aneurism, of this phenomenon; and a spanæmic state of the blood contributes greatly to intensify it, when other conditions are favorable. Thrill may, in such a combination of circumstances, be felt two or three inches beyond the limits of the dilated vessel. Certain morbid states of the blood, especially spanæmia, will suffice, independently of any textural disease, to produce arterial thrill, which, though slighter in amount, is generally more diffused than that of organic origin.

Thrill may sometimes be produced by pressure on a vessel beyond, or on the distal side of, the point pressed on—being imperceptible at the spot which is actually impressed by the finger.[1]

259. The mode of examining the arterial diastole, as of the cardiac systole, by the application of a finger or fingers, is that which has prevailed in all countries ever since the pulse was first examined. The difficulty of comparing observations on strength has, however, from time to time led to the invention of instruments designed to express, by metrical notation, the varying force of impulse. The sphygmometer of M. Hérissant, invented in 1834, was ingenious enough;—but, as I found in using it, error is as likely to arise from the varying manner of applying the instrument (a membrane gives support to a column of mercury in a graduated tube), as from the varying manners of applying the finger in the ordinary way. Whether the sphygmoscope, or cardioscope, recently introduced by Dr. Alison, be less likely to deceive, remains to be seen.

SECTION III.—PERCUSSION.

A.—IN HEALTH.

260. In the natural state, the thoracic aorta has no appreciable influence on the percussion-sound of the surface, beneath which it lies. Even when the arch lies high, the resonance at the sternal notch can scarce be said, practically speaking, to be affected.

B.—IN DISEASE.

261. But when the vessel is notably dilated, dulness under percussion of course occurs, proportional in extent and intensity to the amount of enlargement—modified in some degree by the fluid or

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[1] Allen, U. C. H., Males, vol. xi. p. 332. The finger laid on the radial feels no thrill; but, pressure being made by one finger, another, applied to the vessel further from the heart, is distinctly affected by thrill.

solid state of the contents of the dilated spot, and, to a serious degree, by the vicinity of the enlargement to the surface, and the condition of the intervening textures.

The dulness of a thoracic sacculated aneurism is not coextensive with its dimensions. From its more or less globular form, a limited portion only of the sac reaches the chest-walls, and the observer cannot safely percuss with sufficient force to detect its deeply-seated parts. Hence, practically, an intra-thoracic, and even an abdominal, aneurismal sac, is always larger than the results of percussion would indicate. In non-saccular dilatation of the aorta, percussion is, for obvious reasons, a surer guide to the size of the enlargement.

The resistance of a sac filled with fibrin is highly marked; and the deficiency of elasticity so peculiar as to help in distinguishing the dulness under percussion from that of other states; as, for instance, tuberculization of adjoining lung. The line of union of simply condensed lung and a saccular dilatation is, however, with much difficulty to be drawn, even with the help of this peculiarity.

It is difficult to fix the smallest amount of dilatation of the arch of the aorta that can be clinically demonstrated. Much will depend on the pains taken in the investigation; much on the thinness of the chest-wall and the healthiness of other parts; but much more on the precise site of the dilatation. In a case where the dilatation increased the width of the vessel, when cut open, by two inches, and hence increased the diameter of the unopened vessel by only about two-thirds of an inch, the fact of its existence, suspected from thrill and undue impulse, was *proved* by percussion. But here the right angle of the arch, the point where the vessel nears the surface most closely, was affected. So small an amount of dilatation could not have been detected in any other part of the arch.[1] Physical evidence will always be difficult to obtain, where a small sac is situated at the posterior surface of the vessel; but by careful percussion in the course of the arch, a sac as large as a good-sized walnut may be discovered, if it lie anywhere between the second right interspace and the left border of the sternum, and there be no special and unusual source of difficulty in the way.

SECTION IV.—AUSCULTATION.

A.—IN HEALTH.

262. *Arterial sounds.*—Two sounds, synchronizing with the systole and diastole of the heart, are, in the state of health, clearly audible in the course of the pulmonary artery and arch of the aorta: gradually weakening in force in the thoracic division of the latter vessel, they are with difficulty caught in the lumbar region, but by depressing the abdomen with the stethoscope, may readily

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[1] H. Gordelier, U. C. H., Males, vol. iii. p. 331, and Consumption Hospital, vol. i. p. 56: the patient was extremely thin, too; an accidental aid of no mean importance.

be heard in front. In some persons, however, a single sound, synchronous with the systole of the heart and diastole of the vessel, can alone be detected below the chest.

In the carotid and subclavian arteries almost always, in the axillary and femoral in a fair proportion of cases, the sound continues double; in vessels more remote from the heart, a single sound only, synchronous with the arterial diastole, can be heard. The more active the circulation, the larger the vessel, the thicker its walls, provided their elasticity be not impaired, the more marked is the sound perceived on auscultation. It is not unusual to find a single, very rarely a double, sound even in the brachial and radial arteries, where these conditions are favorable.

263. In the vicinity of the heart, the arterial sounds closely resemble the cardiac, in quality, pitch, and proportional duration; at a short distance from the heart, both become equalized in length and loudness; sometimes in the carotids the second is the longer and louder of the two. At any distance from the heart the sound loses tone completely.

If the vessels are full, and the blood of natural composition, the arterial sound is duller, but more prolonged, than when the mass of blood is small, and its quality thin. The arterial sounds are louder in females, children, and thin persons, than in males, adults, and stout people. Their intensity is increased by slight pressure; a blowing murmur takes their place, in a large proportion of persons, under strong pressure.[1]

264. The arterial sounds heard in the vicinity of the heart are, doubtless, in great part transmitted from that organ; as the second can rarely be heard at any distance from the heart, the inference indeed arises that it is solely a transmission sound, and that the systole of the arteries, at least of the minor ones, is noiseless. But that the impulsion and friction of the blood against the vessels, and vibrations of their walls, during their diastole, with the current-like motion given to their column of blood by the form of the vessels, generates sound, cannot be doubted; it is not conceivable that sound, audible in the popliteal or radial artery, is the mere result of conduction from the heart. Besides, it is not very uncommon to find the first sound in the carotid artery, oftener in the right than the left, stronger than at the aortic orifice.

B.—IN DISEASE.

265. The natural amount of arterial sound is capable of increase or diminution both in the aorta and pulmonary artery.

But the only modification of clinical significance, falling under this head, is reinforcement of the second sound in the pulmonary artery—a state connected by Skoda directly with hypertropy or excessive systolic action of the vessel itself, indirectly with mitral constriction and regurgitation, and with hypertrophy of the right ventricle [211].

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[1] But it is an error to suppose that any amount of pressure will produce murmurs in all persons and in all vessels: the contrary may be hourly verified at the bedside. The smaller the artery the more easily is murmur generated.

266. *Arterial murmurs.*—The arteries, like the heart, become the seat of murmurs, either from organic change or independently of this. They may be arranged as follows:—

1. Organic.
2. Inorganic.
   - (*a*) Hæmic.
     - From altered composition.
     - From coagulation or deposition.
   - (*b*) Dynamic.

267. *Organic* murmur in the intra-thoracic arteries varies in intensity from a scarcely perceptible sound to one audible even at a slight distance from the surface; is limited to a small portion, or, more rarely, extends over a considerable tract, of the vessel; presents all the varieties of quality noted in cardiac murmurs; varies in pitch from that of the word *awe* whispered in inspiration, to a high whistling note; is of distinctly intermittent rhythm, single, either systolic or diastolic, or double; and either short and abrupt, or prolonged and slow.

268. The organic conditions of arterial murmur are referable to (1) change of form of the vessel; (2) to a modified condition of its internal surface; (3) to both these states combined; and (4) to communication between an artery and some portion of the venous system.

269. (1.) Peripheric dilatation, or simple lateral sacculation of an artery, especially if abrupt and well-marked, by altering the direction of the blood-current, generates murmur, even if the internal surface be perfectly smooth. Narrowing of a vessel produces a similar effect, by increasing the friction of the blood against the walls at the constricted point.[1] Murmur of both these kinds is always synchronous with the pulse, and not rough, unless the blood be spanæmic, and the inner walls deficient in smoothness.

270. (2.) Mere atheroma, unless accumulated in unusually large quantity, does not produce murmur; infiltration with induration-matter, by puckering the inner surface irregularly, roughens the sound; erosions of the lining membrane do so more effectually; and calcification of the vessel, if it cause irregular elevations of that membrane, is still more efficient. Murmur is also produced by particles of exudation-matter studding the surface of an artery recently inflamed. This variety of murmur is always synchronous with the pulse.

271. (3.) In the varieties of aneurism with diseased coats, form and surface are changed so as to produce murmur, which may be

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[1] Dr. Corrigan, however, argues with great ingenuity, that it is not at the constricted point of a vessel, but within the area inclosed by the loose walls beyond, that murmur is produced; and the fact mentioned above [258] concerning thrill beyond the point of compression of a vessel would support his view.

systolic, diastolic, or both, and attended or not with sound also. The conditions regulating the time and accompaniments of this murmur will be described under the head of Aneurism of the arch of the Aorta.

272. (4.) Intra-thoracic varicose aneurism, in all its forms, is attended with murmur synchronous with the arterial diastole, sometimes prolonged through its systole.

273. A certain amount of force in the heart's action is essential to the generation of arterial murmur; increase of that force will convert a soft into a harsh quality instantaneously; again, a well-marked murmur will sometimes disappear under the influence of slight faintness, falling far short of actual syncope.

274. Murmurs heard in the thoracic aorta, single or double, are often merely conducted from the heart. But if a murmur, audible at any part of the arch, be of notably different pitch, of greater intensity, and of harsher quality than a synchronous murmur at the aortic base, it may be inferred that a cause of reinforcement exists in the vessel itself. The only source of fallacy would be the chance coexistence of badly conducting materials over the base of the heart, and of excessively good ones over the arch of the aorta. The characters of an arterial murmur will sometimes guide the observer partially to a knowledge of its anatomical cause; but the actual determination of this will mainly turn on the state of other physical signs [157].

275. *Inorganic murmurs.* (*a.*) *Hæmic.*—(1.) *From altered composition.*—Inorganic arterial murmur of this description is commonly softly blowing, if the vessel be auscultated without pressure. If pressure be used, it rises in pitch, and becomes sharply whiffing or whipping, resembling the sound produced by a quick stroke of a riding-whip through the air. As a rule it is intermittent, single, never continuous, synchronous with the diastole, and never solely with the systole of the vessel, and affects the arterial system extensively, instead of being purely local, as the organic variety.

In rare instances, this variety of murmur is distinctly double in vessels lying within the ready influence of pressure; I have known it so in the femoral artery, for instance—here the portion corresponding to the systole of the vessel may exceed the diastolic in length.[1]

The clinical conditions of this form of inorganic arterial murmur are certain of those of cardiac murmur of the blood-class, especially spanæmia. It is said that plethora produces it—a statement I have been unable to verify clinically. But of this more by-and-by.

276. Midway between the organic and inorganic varieties stands the murmur of an artery, healthy in itself, but pressed upon by an

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[1] Gall, U. C. H., Males, vol. xvii. p. 272. Here there is powerful systolic murmur at the aortic orifice, but none of diastolic rhythm.

adjacent tumor. Arterial murmur of this mechanism is commonly synchronous with the pulse, that is, diastolic in rhythm, in regard of the vessel. But, just as may sometimes be observed in arterial murmur artificially produced by stethoscopic pressure [263], so, in the case of tumor-pressure, the murmur may be double, synchronizing both with systole and diastole; and this in the case of a vessel beyond the reach of influence by the stethoscope. I have substantiated the fact, for instance, in the abdominal aorta, when ausculted in the lumbar groove;[1] its importance in regard of the diagnosis of aneurism is obvious.

277. (2.) *From coagulation or deposition.*—Slight roughnesses on the inner surface of an artery, probably insufficient in themselves to produce such effect, lead indirectly to the generation of murmurs by causing local deposition of fibrin.

Coagulation occurring on a large scale must directly generate murmur. I believe that I have heard murmur of this mechanism, extending outwards on both sides of the sternum, and apparently following the course of the pulmonary artery, in a case where life was brought to a rapid close by coagulation within that vessel.

278. (*b.*) *Dynamic murmurs.*—That dynamic influence on the part of the arterial walls plays an important part in certain diastolic (that is, systolic as concerns the vessel's rhythm) aneurismal murmurs, cannot be doubted: the reaction of the sac on the distending current, sent on by the heart's systole, is sufficient to disturb the flow so as to produce murmur.

It is possible, too, that some such influence may be at work in the cases of double inorganic arterial murmur a moment since referred to.

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[1] Gosling, U. C. H., Females, vol. v. p. 130.

§ III.—VENOUS SYSTEM.

SECTION I.—INSPECTION.

279. It is not intended here to refer to the signs of *local* diseases of the veins, such as varix or phlebitis. Now, such local changes being excluded, it follows that considerable distension of any particular portion of the venous system indicates the existence of obstruction in the connected main trunk, or in the right side of the heart itself.

280. The internal and external jugular veins are the veins most frequently found enlarged—the right more commonly than the left, when one side only is affected. This obstructive distension, uniform or varicose, even if increasing the size of the external jugular almost to that of the little finger, is unattended either with change in the integuments, hardness or cordiness of the vein, or tenderness under pressure. The common cardiac cause of this condition is tricuspid regurgitation; a more rare one, simple dilatation of the right cavities of the heart: beyond the heart it depends on pressure on the superior cava, or innominate veins by intra-thoracic tumor or aneurism. So that in the one case it comes of too free, in the other of obstructed, communication between the cervical veins and the right auricle. Another infinitely rare cause of the phenomenon is aneurismal varix within the thorax.

Excess of enlargement on one side cannot be trusted to as significant of local pressure, instead of cardiac disease, as the cause of enlargement. Dilatation of the heart and tricuspid regurgitation may be attended with excess of enlargement of the left, as well as of the right, external jugular; such excess is indeed common on the right side, rare on the left.[1]

Jugular distension is very sensibly affected by the patient's posture: the nearer the head and neck are placed on the level of the heart, the greater the fulness—insomuch that a greatly widened vessel in the ordinary recumbent position may, in the sitting attitude, cease to be of sufficient size to attract attention. As the patient turns from side to side, something of the same kind may occasionally be witnessed: thus, in a case where in dorsal decumbency the left external jugular was notably the fuller of the two, it became sensibly the emptier when the patient turned on the right side.[1]

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[1] Couch, U. C. H., Females, vol. vii. p. 344.

281. The external jugular vein, oftener the right than the left, is occasionally the seat of visible pulsation, especially at its lower part near the clavicle. Irregular in amount and in rhythm, though obviously connected in the main with the ventricular systole, jugular pulsation wants the distinctness of an arterial pulse, and is rather an unsteady intermittent tremulousness than a series of well-defined beats. The effect of inspiration and expiration on the blood in these veins partly explains the irregular rhythm; which may also, in part, be referred with extreme probability to the influence of the auricular systole. The impulse producing it comes visibly from below: and when a portion of the vein is emptied by pressure from the clavicle in an upward direction, it refills immediately from below, while the pressure is sustained above.

The amounts of enlargement and of pulsation are not directly proportionate: oftentimes indeed the reverse. There is a point of extreme distension at which the vessel is perfectly motionless.

Lancisi, the original observer of this phenomenon, supposed that it was produced by eccentric hypertrophy of the right ventricle. Hope, holding to this view, explains the impulse by the "impetuous recoil of the tricuspid valve," which repels the blood about to descend into the ventricle with such force that its impulse is propagated back to the jugular veins. Many persons maintain that jugular pulsation only occurs where the tricuspid orifice is too much dilated to admit of closure by its valve, whence ensues regurgitation into the veins during the ventricular systole. Dr. Parkes teaches that, in addition to tricuspid insufficiency, rupture of the valves at the junction of the internal jugular and subclavian veins is a necessary condition of the phenomenon.

I know of no facts positively showing the necessity of such rupture of valves; the vessels may be sufficiently distended to render their valves incompetent, which is all that is required.[2] The valves, too, may be congenitally absent. Further observation on these points is, however, desirable. But, as concerns the tricuspid orifice? Unquestionably jugular pulsation is most frequently met with in cases of tricuspid insufficiency, though by no means in all of the class; while, as I have decidedly observed it, where the valve was not demonstrably incompetent, in cases of dilated and hypertrophous right ventricle, I cannot refuse to admit that this latter condition alone may produce it.[3] I believe, too, slight pulsation may occur in pure palpitation, disappearing with the cessation of the fit. If the ventricle be hypertrophous, and the valve insufficient, the pulsation reaches its maximum. It is to be remembered, too, that respiration affects jugular pulsation, emptying the vein in inspiration, distending it in expiration. The parts played severally by respiration and cardiac action, may be distinguished by causing the patient to suspend his breath for a moment.[1]

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[1] Couch, U. C. H., Females, vol. vii. p. 345.

[2] Thos. Denham, U. C. H., Feb. 1851, Males, vol. vi. pp. 69, 77. Here notable pulsation, both of the jugular and innominate veins, had existed during life; the valve was ascertained to be perfect, but enlargement of the calibre of the veins had obviously rendered it incompetent.

[3] It is too constantly assumed by observers, that where the tricuspid valve is insufficient to close the orifice at death, it has been so during life also. What proof have we that an instinctive constriction of the orifice does not, except in

Extreme jugular pulsation may exist without the least visible throbbing of any other vein.

282. The right mammary veins may be knotty and pulsatile; I have seen this in cases of insufficiency of the tricuspid valve, and of intra-thoracic tumor.

283. But veins, much more distant from the heart than these—the veins, for instance, of the *dorsa of the hands and feet*—may be the seat of pulsations either of cardiac or of respiratory rhythm, or of both combined. Dr. Jenner has very kindly favored me with the particulars of three cases illustrating these various rhythms. When the rhythm is cardiac, pressure on one of the pulsatory veins on the back of the hand increases the strength and distinctness of the pulsations (which equal in number those of the radial pulse) to the distal side of the point pressed on, annuls them to its proximal side; the respiratory movements exercise no influence on the pulsations. When the rhythm is respiratory, the vein collapses in inspiration rapidly, swells in expiration slowly; and, when pressed on, its pulsation ceases to the distal, increases to the proximal, side of the point pressed on. One of Dr. Jenner's cases exhibits the coexistence of the two sorts of pulsation (respiratory—and cardiac, by *vis à tergo* through the capillaries, probably) in an infant aged eighteen months, cut off with pneumonia secondary to tubercles. The reason why respiration and cardiac action should exercise this influence on distant veins in some cases of disturbed thoracic action, and not in others, seemingly similar, is yet to be discovered.

284. In all cases of apparently intrinsic venous pulsation, the possible influence of contiguous arteries must be borne in mind.

SECTION II.—APPLICATION OF THE HAND.

285. Thickening of the walls of the jugular veins sometimes arises in cases of tricuspid regurgitation of long standing. If in such a case those vessels pulsate, it is very probable their diastole

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extreme cases, accommodate the width of the opening to the capabilities of the valve?

[1] The jugular pulsation sometimes noticed in pneumonia may depend either on cardiac influence, or on that of respiration and the pulsation of the contiguous artery combined; the fluttering movement of the vein on the affected side sometimes attending pleuritic effusion is, as far as I have seen, of the latter mechanism, and may totally disappear in the erect posture, though well marked in decumbency (I. Harrison, U. C. H., Females, vol. ix. pp. 329, 345).

will be perceptible to the fingers; but I do not remember ever to have actually observed this.

286. Visible pulsation is in some instances attended with soft thrill—a minor degree of the arterial phenomenon of the same name. The most marked example is furnished by that excessively rare diseased state, aneurism of the arch communicating with the vena cava.

SECTION III.—AUSCULTATION.

287. It was long believed that the blood-current in the veins is, in the normal state, non-soniferous. Observations have, however, gradually accumulated of late years showing conclusively that in a large proportion of persons possessing all the attributes of the ruddiest health, venous murmur may be detected—occasionally even in veins where no pressure with the stethoscope could interfere with the flow of blood within the vessels examined. In order to avoid needless repetition, I defer to a future paragraph an epitome of facts bearing on this part of the subject [294].

288. *Characters of venous murmur.*—Murmur, occurring in the veins, possesses one invariable character, that of continuousness, uniform or remittent.

289. In point of quality, venous murmurs are referable to four types: the blowing, the whistling, the humming, and the modulated. The blowing varieties may be as soft as the respiration-sounds in health, sharply blowing, loudly blowing, as the sound heard on applying a shell to the ear, or actually roaring. Or, the murmur may be cooing or whistling. To the humming type belong various murmurs resembling more or less closely the noise of a humming-top, the buzzing of a fly, the singing of a tea-kettle, &c. Lastly, venous murmurs are sometimes distinctly modulated, consisting of a series of separate tones, capable of musical notation, recurring at tolerably regular intervals, and accompanied by a low hum, which gives the continuous character to the whole.

Inclining rather to softness than roughness, and of moderate intensity, inaudible unless the ear or stethoscope be applied directly to the surface, generally of low pitch, as the whispered word *who* (when modulated, of course, this is variable), venous murmur is liable to change in intensity and quality from one moment to another. This change occasionally occurs from some intrinsic untraceable agency; more frequently from some one of the following causes. Acceleration of the circulation intensifies venous murmur; and as inspiration favors the rapid flow of blood in the veins adjoining the thorax, in these veins, at least, that act ought to, and does actually, increase the loudness of an existing murmur. But, on the other hand, suspension of the breath at first exercises even more markedly the same effect; the sharp collision of the blood disks *inter se*, and against the walls in the struggle to move onwards, and the increased vibration of the walls themselves, probably explains this. If the breath be held for any time, the murmur disappears. Any posture which stretches moderately the vein under examination, intensifies its murmur: if the part examined be a muscular one—the thigh, for example—there is a source of fallacy in the rumbling sound of muscular contraction, which must be guarded against by auscultation in a perfectly relaxed condition of the muscles. In the neck, murmur is stronger in the erect than in the lying posture; doubtless from the greater rapidity of flow in the former attitude. Venous murmur attains its maximum under a certain amount of pressure, ascertainable in each instance only by actual experiment. Less or more pressure weakens and finally obliterates all audible sound. Sex exercises no influence on the intensity of venous murmur, nor, directly, on its frequency. No doubt, it is greatly more commonly observed clinically in females than in males; but this depends simply on the disproportionate frequency of its physical conditions in the two sexes. No evidence has ever been adduced showing that a given state, which fails to generate murmur in a male, will succeed in the case of a female.

290. The veins in which murmur occurs, may, as far as I have observed, be arranged as follows in order of frequency. The external and internal jugulars, on both sides, or on one side only, in the latter case most frequently the right; the subclavian veins; the femoral (I have never failed to find it in these vessels when well developed in the neck, and it may be caught in them sometimes when inaudible in the jugulars); the axillary, the superior cava and innominate veins;[1] the veins of the bend of the elbow; certain abdominal veins;[2] the pulmonary veins; and the superior longitudinal sinus, especially at its termination in the torcular Herophili.[3]

291. Invariably continuous and never intermittent in rhythm, murmur in a vein may be simply continuous, that is, of equable force constantly; or it may be remittently continuous, undergoing reinforcement and weakening at regular intervals. The type is humming when the rhythm is thus remittent. Two causes of this remittent character have been suggested; the pulsations of an adjoining artery against the soniferous vessel, which are supposed to give, at regular intervals, a momentary impetus to the current in the interior of the vein; and the coexistence of ordinary intermittent blowing murmur in the accompanying artery. The majority of instances are fairly explicable on one or other of these principles;

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[1] M. Clarke, U. C. H., Females, vol. iv. p. 261, Oct. 1848. "Lancet," March 31, 1849.

[2] Sus. Roberts, U. C. H., Females, vol. v. pp. 218–220, Oct. 1850. Continuous loud "roaring" hum, coupled with arterial intermittent murmur, of maximum strength a little above and to the *left* of the umbilicus, but audible even to the right of the middle line. Here, too, appear cases of continuous hum, audible on deep pressure at the right edge of *some* enlarged spleens.

[3] Davis, U. C. H., Females, vol. iv. p. 138. In this case a continuous remittent murmur was also audible at both sides of the mid-dorsal spine.

some, which are not so, may, it would appear, be explained by intrinsic inequality of force of current in the veins—an inequality which, we know, positively exists in cases of venous pulse.

292. Venous murmur may be accompanied or not with arterial or cardiac murmur, the latter basic in seat and systolic in time.

293. But do the murmurs, just described under the name of venous, really deserve the title? The history of opinion on the point is exceedingly curious. Long after the ingenious and logical inquiry of Dr. Ogier Ward had demonstrated the venous site of the cervical "humming-top" murmur to the satisfaction of observers in this country, M. Bouillaud, and French writers generally, persisted in localizing it in the carotid arteries. Skoda, treating of it in 1842 in his section on arterial murmurs, likewise placed it in the carotid, and actually ridiculed the idea of venous origin.[1] And now that he has adopted the correct view, his countryman, Kiwisch, experimentalizes and writes to prove that the veins are never the seat of murmur, the carotids being the real site of the cervical hum in a chlorotic woman. Kiwisch's argument, however, seems to me simply to present one more illustration of the unfortunate facility with which, in matters medical, plausible ingenuity may succeed in the effort to make "the worse appear the better reason." The following, among other facts, appear, in truth, conclusive as to the venous origin of these murmurs.

1. The gentlest pressure on the external jugular above the point on which the stethoscope rests, instantaneously silences the hum beneath—an amount of pressure so slight as to be incapable of exercising the least influence on the neighboring artery. 2. Continuous hum and either arterial sound or arterial intermittent blowing murmur may in some cases be heard at one and the same moment separately; I have repeatedly noticed this in the femoral artery and vein. 3. Continuous hum can be heard where there is no artery to furnish murmur—as, for instance, along the longitudinal sinus and at the torcular Herophili. 4. The erect posture increases, the lying posture lessens, continuous murmur in the neck; the precise converse effects are observed below the groin; this is only intelligible by the changes in the venous current produced by altered postures—altered postures which exercise no appreciable influence on arterial action. 5. Continuous murmurs of different quality and different pitch can occasionally be heard over the external and internal jugulars; there is no difference of artery in the two situations.[2]

294. Various hypotheses suggest themselves in explanation of these murmurs, referring them severally to altered composition of the blood, diminution of the mass of the blood, change in the

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[1] Percussion, &c., 2te Aufl. p. 199.

[2] Perhaps it is not altogether unworthy of notice, also, that Kiwisch failed to produce continuous murmur in the arteries laid bare, and auscultated under various amounts of pressure in large-sized animals.

blood's motion, and change in the dynamic and physical conditions of the walls of the veins.

(*a*.) Venous murmurs are so constantly connected clinically with chlorosis and other maladies, of which poverty of blood forms an element, that their generation in some way or other through that condition of blood was generally, and without, it must be confessed, actual proof, accepted as a fact. The deficiency of red disks in anæmia, hydræmia, and spanæmia was held to explain the noisy movement of the blood—a notion which received its first contingent of seemingly precise support from M. Andral, who, attempting to establish the exact relationship between the amount of spanæmic change and the constancy of venous murmur, finds that if the red corpuscles fall below 80 per 1000, murmur is constant; if they range between 80 and 100, pretty frequent; if between 100 and 115, occasional; if between 115 and 126, very rare; if they reach the average of health, invariably absent. It is argued that the blood being unnaturally thin, the friction attending its movement is, according to a law of Poisseuille's, increased to such an amount as to engender sound.

But there are some facts obviously inconsistent with this theory, at least as an exclusive one. Thus it is well known that in cases of chlorosis, treated with iron, color returns to the tissues long before venous murmur disappears; and, *vice versa*, Becquerel and Rodier give analyses of the blood of two chlorotic girls, presenting well-marked venous hum, with a mean proportion of 125.1 per 1000 of red corpuscles—certainly an amount falling within the limits of health. It was long since stated, too, by the "London Heart Committee," that murmur may be produced in the veins by pressure in a state of robust health; and I have heard it in women of florid complexion, who certainly were not at the time of examination (and as far as I could ascertain, had never been) *symptomatically* anæmic.

Gradually observers grew prepared for the announcement, first, as I believe, made by Liman, that the venous current is soniferous, under the ordinary circumstances of stethoscopic examination, in a large proportion of children and adults totally free from appreciable disease. Many persons have subsequently gone over the same ground as Liman, and the following table, contributed by Winterich,[1] fairly represents the general results in *healthy* individuals.

PERCENTAGE OF VENOUS MURMUR IN HEALTH.

Age.	Males.	Females.	Age.	Males.	Females.
1 — 5	97	98	30 — 40	80	86
5 — 10	94	95	40 — 50	77	78
10 — 15	89	95	50 — 60	72	75
15 — 20	86	88	60 — 70	68	71
20 — 25	82	88	70 — 80	40	39
25 — 30	80	86			

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[1] British and Foreign Medico-Chirurgical Review, April, 1852. The inquiries of Dr. H. Davies may also be referred to.

From this table it follows that normal venous murmur, almost constant in early infancy, gradually decreases in frequency with advancing years, attaining its minimum of about 40 per 100, in the decennial period 70 to 80. The average frequency of the murmur is slightly greater in females than males.

But, as far as my own observation goes, these returns do not wholly disprove the importance of spanæmia as an element of venous hum; for I have never yet succeeded in finding a strongly marked murmur of the kind in healthy people. The elements of the phenomenon in a minor degree exist in health; but in some way or other the clinical conditions of spanæmia are connected with its highly developed forms. There is a *normal* and an *abnormal* amount.

Is it possible that increase in the proportion of white corpuscles, a very positive attendant on chlorosis, and, as shown by Remak, on repeated hemorrhage, plays a part in generating venous murmur—such increase probably entailing increase of friction and labor in the circulation? It is certain that with disappearing anæmia and lessening hum the white corpuscles may be found to diminish;[1] but of course association does not prove causation. And I must confess my failure to discover any high degree of hum in certain cases of leucohæmia would tend to invalidate this view.

(*b.*) Pure diminution in the mass of the blood explains jugular hum according to Hamernjk, by the whirling and eddying motion into which the dwindled venous current is thrown in passing along a vessel, which in consequence of its anatomical arrangement always retains a certain width at its lower part. But it seems impossible to prove this alleged influence of anæmia to the exclusion of spanæmia, inasmuch as diminution of the blood's mass seems never to take place without a fall in the proportion of red corpuscles.

(*c.*) The influence of eddying and interrupted motion, however, seems difficult to contest in face of the manifest part played by regulated pressure with the stethoscope in eliciting the murmur.

(*d.*) If the veins contain less blood than natural, their walls become looser—the increased facility of vibration, thus arising, accounts for the murmur in the opinion of some. M. Vernois, in a modified form of this theory, looks on the prominent folds, into which the walls of the veins are thrown, as the cause of murmur, through the impediments they offer to the flowing blood. But as the sinuses of the dura mater, with their quasi-rigid walls, give murmur, the non-necessity of such obstructive action is obvious.

(*e.*) Experiments have been made on fluids traversing tubes by M. Weber, tending to show by inference that the vibrations of the walls of the vessels themselves, rather than of the contained fluid against those walls, cause the audible murmur.

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[1] M. Pitt, U. C. H., Females, vol. viii. pp. 21, 57, Nov. 1851.

(*f*.) The theory referring the sound to contraction of the platysma myoides is the feeblest that has yet been advanced. How comes it, if such be the mechanism of venous hum, that sound is heard only over those parts of the platysma corresponding to the jugulars—and that it may be caught in situations where no muscular substance exists at all?

295. The real mechanism of these murmurs is consequently obscure enough. It seems to me impossible to ignore the influence of the composition of the blood; in highly marked spanæmia, neither flaccidity of veins, pressure on their surface, nor even velocity of current, is required for the generation of murmur—it exists in the sinuses of the dura mater, especially where the arrangement of these canals is such as to promote abrupt collision of opposing currents meeting at a conflux, as at the torcular Herophili.

296. It is held by some writers, that as murmurs may be heard in the veins of healthy-looking persons, they cannot be accepted as evidence of a morbid state of the blood. Such "normal" venous murmur, as already observed, is relatively very feeble in the adult. And, inasmuch as venous murmur cannot be found by any means in all adults, there is clearly something not perfectly natural in those who present it. Even in children it cannot invariably be caught; and what is healthy in a child may be morbid in an adult —take the single instance of the different states of Peyer's glands in childhood, and more advanced life. The composition of the blood differs in infancy and adult age; it acquires new characters, again, in old age, and these characters may very possibly render the fluid less apt to generate sound, whence the less frequency of venous murmur in old age.

I would sum up on this matter, clinically, by saying, that a state of blood in which the venous current becomes slightly soniferous, is not incompatible with apparently perfect health; but that intense, readily audible and diffused venous murmur, is characteristic of those morbid conditions of the fluid, called hydræmia, anæmia, and spanæmia.[1]

297. The diagnosis of venous murmurs turns essentially on their continuous character; and is excessively easy, except when accidental circumstances occur to render that character obscure. This happens sometimes about the base of the heart anteriorly, and between the scapulæ in the back. Pulmonary venous murmurs are partially masked by the cardiac sounds.[2] In addition to its peculiar quality, pitch, inconstancy, ready influence by change of posture, as guides to the venous origin of the murmur, its rhythm in respect of the heart's beat will *sometimes* aid in connecting it with

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[1] The importance of this state of the fluid seems, indeed, to have received the final demonstration by the experiments of M. Weber, who was shown that murmur is much more readily produced in tubes if an admixture of blood and water, than if pure blood, be used as the moving fluid. (Ranking's Retrospect, vol. xxiv.)

[2] Commentary on case of Clarke, "Lancet," March, 1849, p. 332.

the veins. Thus a diastolic murmur at the base (the signs of *organic* disease at the cardiac orifices being deficient) must be venous, according to my experience;—at least an inorganic cardiac murmur of that site and rhythm has never fallen under my notice.

298. The occurrence of murmur in cases of intra-thoracic varix has already been mentioned: on the same principle, if a lateral communication exist in any part of the body between a contiguous artery and vein, so as to permit the current from the former to enter the latter, a murmur of whizzing or whirring character, and essentially intermittent, results, mingled with a continuous sound, specially engendered in the vein.

299. It is conceivable, too, that a murmur of systolic rhythm only, and consequently intermittent, shall be produced within the superior cava, and at the line of union of this vessel within the innominate veins in cases of highly marked tricuspid regurgitation. But I am not sure that I have ever heard murmur of this mechanism.

AUTO-AUDIBLE MURMURS.

300. Although our knowledge on the subject be as yet limited, it may prove not without advantage to group together such facts as have been established concerning murmurs audible by the individual himself in whose frame they occur. Such murmurs, which we may distinguish as *auto-audible*, may be venous, arterial, endo- or pericardial.

301. That murmur in the cervical and connected veins is more or less distressingly audible in cases of anæmia, producing various forms of "singing noise" in the ears and head is well known. So, too, whiffing intermittent murmurs in the carotid, especially in recumbency and on the side on which the patient happens to lie. A remarkable case is referred to in the description of aneurism of the aorta, in which murmur, not only auto-audible, but distinctly perceived by the bystander at a certain distance from the body, was heard through a considerable portion of the arterial system.

302. Patients, the subjects either of organic or functional cardiac disease, will occasionally describe noises, referred by them to the heart, and in which the physician at once recognizes the characters of murmurs. Under these circumstances, I have actually known endocardial murmurs exist of two kinds; systolic at the base, and systolic at the left apex. In the former of these cases, the explanation seems easy; the murmur, transmitted along the aorta, reached the carotids. But in the latter case of mitral regurgitation, the explanation is not so readily forthcoming. In one instance of the kind, where there was neither carotid whiff nor jugular hum, but the mitral murmur was strongly reinforced by a flatulent stomach, I cannot refuse to believe that the patient actually heard that murmur, as she described its characters accurately, and, her heart never having been physically examined, until she was seen by myself, she could not, as it were, have learned her lesson from others.

303. Lastly, I have lately seen a case in which pericardial high-pitched clicking sound was distinctly heard by the patient: the sound, audible through the stethoscope, so precisely agreed in its characters with the description given by the patient of the peculiar noise he heard, that no doubt could be entertained on the question. But I am unable to explain why murmur in the pericardium (it wholly disappeared both to observer and patient with the disappearance of acute symptoms) should have been auto-audible in this particular case.

PART II.

DISEASES OF THE HEART AND GREAT VESSELS.

DISEASES OF THE HEART.

304. DISEASES of the heart, as of all other organs in the body, are divisible into two classes: in the one, as far as can be discovered, the dynamics of the organ alone are at fault; in the other, structural change is more or less apparent. Cardiac affections are hence dynamic and organic.

SECTION I.—DYNAMIC DISEASES OF THE HEART.

305. The different perversions of the dynamics of the heart which are known clinically, may exist in association with structural disease, as well as independently of this. The distinction of the two conditions—the associated and the unassociated—sometimes simple enough, often proves in actual practice far from easy.

306. A review of some of the more important of the alleged rules for distinguishing the two classes will at once show their clinical insignificance. The inconstancy of the symptoms of dynamic, and the constancy of those of organic ailment, are strongly dwelt on, for example; but all the subjective, and many of the objective, symptoms may disappear temporarily in cases even of extensive organic disease. Such disappearance with ensuing recurrence may even take place several times. The existence of secondary changes, such as subcutaneous œdema, and congestion of the lung, commonly proves the cardiac affection to be organic: but not always, for spanæmia, added to nervous palpitation, may induce œdema. If exercise relieve a disturbed heart, its affection is pronounced to be dynamic only; if movement increase the suffering, organic. This proposition might lead to an incorrect impression; for, it is certain, if spanæmia coexist with perverted action, exercise may be unbearable. If, in the intervals of attacks of disturbed action, the force and rhythm of the pulse and heart are natural, those attacks are said of necessity to be functional: an error; for the most perfect tranquillity of the organ *may* exist, from time to time, though

its texture is seriously unsound. It has been taught that inability to bear particular postures, more particularly that of decumbency on the left side, in cases of disordered action of the heart, shows that organic mischief exists. Often true, this proposition occasionally proves false: women with spanæmia, palpitating, though perfectly sound, heart, and left intercostal neuralgia, can rarely endure the posture in question.

307. Singularly enough, the amount of local suffering entailed by disturbance wholly, or in the main, dynamic is often greater—is, in the mass of cases, greater—than that produced by actual organic disease. The patient with grave organic mischief, that may kill him at any moment, say, marked aortic regurgitation, may be so free from cardiac suffering, as to express irritation that his heart should be made the subject of examination: another individual oppressed with disturbed rhythm and innervation of his structurally healthy heart, dependent on flatulent distension of the stomach, refuses to be persuaded of his freedom from mortal cardiac mischief. As a rule, apprehension is not often excited in the subject of structural disease of the heart, until the secondary and distant evils of the local morbid changes, whatever they be, begin to make themselves felt.

308. The truth is, careful and repeated physical examination alone can justify a positive opinion on this question; and even this aid will occasionally fail. For, in the first place, the mere existence of certain abnormal physical signs in a palpitating heart does not warrant the assumption that textural change exists. Thus a basic systolic murmur may be simply anæmic—it may by possibility arise from mere perverted action of the sigmoid valves—or depend simply on the unnatural force with which the blood-current rushes against the orifices of the great vessels. Again, a systolic murmur at the left apex of the heart may be generated by irregular action of the papillary muscles. Further, extension of the normal area of cardiac dulness even considerably to the right may depend on merely temporary obstruction of the blood's movement through the heart, leading to distension of its right cavities, and be wholly independent of any real dilatation or hypertrophy. Conversely, in the second place, the total absence of physical signs does not prove the heart to be in a perfect state of organic soundness: there are slight amounts of change in the heart's substance, of which the perverted signs (for, doubtless, such really exist) are beyond the penetration of the present day. Nor must the student forget that numerous conditions of adjacent structures may throw cardiac physical signs into the shade, and prevent the detection, unless extreme care be exercised, of any positive disease. Thus emphysematous or even bronchitic distension of the lung, causing this organ to overlap the heart to an abnormal extent, will prevent increased area of percussion-dulness by an enlarged heart; and horizontal conduction of amphoric note from the stomach or colon

[99] may gravely deceive as to the actual outline of the heart. And such instances might readily be multiplied.

309. Assistance in cases of doubt may be obtained from diathetic peculiarities and coexistent diseased states. Functional disturbance of the heart is connected more or less constantly and markedly with the following conditions: *Perverted innervation*, as in cases of hysteria, spinal irritation, uterine and ovarian excitement, and various neuralgiæ, intercostal, dental, &c., *altered condition of the blood*, as in hemorrhage, anæmia, gout, chronic rheumatism, and chronic disease of the liver; *nervous exhaustion*, as from sexual excess, masturbation, or spermatorrhœa: *mechanical interference with the heart*, as when the stomach or intestines are disturbed with flatus: *certain poisonous influences*, as those of tobacco, green tea, and various diffusible stimulants. But none of these conditions are inconsistent with the presence of actual organic mischief.

Nevertheless, although cases occasionally occur in which it proves impossible to affirm or deny the presence of organic change (more especially, perhaps, of fatty atrophy), the instances in which a painstaking observer is wholly baffled are really rare.

310. Now functional disorders of the heart, clinically recognized, being composed of various disturbances of the elementary dynamic properties of the heart, innervation and contractility, it seems likely to tend to a better understanding of those functional disorders, if we commence by an analysis of the demonstrable or possible perversions of the elementary properties. Clinically, however, this study is in its infancy; physiologically, even, our information is unsettled and incomplete. Hence, to a considerable extent, the statements I am about to lay before the student are to be accepted rather in the light of suggestion than of dogmatic assertion.

§ I.—ELEMENTARY DYNAMIC CHANGES.

311. These changes affect, as just mentioned, Innervation and Muscular Action.

A.—PERVERTED INNERVATION OF THE HEART.

I.—Cardiac Hyperæsthesia.

312. Hyperæsthesia of the cardiac system of nerves may be supposed to induce neuralgia, to exalt the tactile sensibility of the organ, and to affect similarly its special sensibility. Let us examine these possible changes seriatim.

(a.)—NEURALGIC HYPERÆSTHESIA.

313. Patients occasionally seek advice for the relief of pain, the chief seat of which appears to correspond to a limited spot about the left fourth intercostal space and fifth rib (sometimes a little higher than this), slightly outside the vertical nipple-line. They maintain, with as much confidence as can reasonably be looked for concerning a problem so difficult as the precise localization of pain, that the suffering in question is situated at a certain, but not great, depth below the surface. Remaining limited to the spot indicated for a variable term, the pain may eventually extend to the upper part of the front chest, reach the left shoulder and base of the neck, extend downwards to, and not beyond, the elbow, or reach the tips of the fingers. Various other modes of radiation of this pain may occur, which will be more fully examined under the head of Angina Pectoris.

314. The amount of suffering ranges between a sensation of little more than uneasiness to one of the keenest anguish. In character the pain may be shooting, pressing, constrictive, or rending.

315. This pain is not a form of intercostal neuralgia: the three characteristic points of hyperæsthesia in the course of the intercostal trunk, at the vertebral groove behind, close to the sternum in front, and midway between those spots, are wanting. Neither is it seated in the cutaneous nerves; as a rule, there is no morbid sensibility of the skin in the spot—far from this, a sense of relief is frequently experienced from gentle and regulated pressure. Nor has it any other than a merely accidental connection with hysteria; it is not only rare in hysterical women, but in the female sex generally.

The same description, *mutatis mutandis*, will apply to a pain experienced occasionally behind, or a little to the right of, the lower part of the sternum in persons free from dyspepsia, pulmonary or hepatic disease, but obviously the subjects of obscure cardiac mischief.

There can be little doubt that the neuralgia described is really seated in the sensory filaments of the cardiac plexus. And this excited sensibility may exist in an isolated condition, no motor disturbance of the heart ensuing—or it may be associated with, or prove the excitant of, disturbance of motor innervation also, leading either to increased or diminished action.

316. Neuralgia of the cardiac plexus is less common where obvious organic disease of the heart exists, than where the evidence of such disease is wholly wanting or obscurely defined. It constitutes in its extreme degrees an essential element of angina pectoris; and with the account of this disease the origin, mechanism, and treatment are considered.

(*b.*)—TACTILE HYPERÆSTHESIA.

317. The heart does not appear to be normally endowed with any high degree of common tactile sensibility. I draw this inference from the positive and negative results of vivisections; and from the only case of wound of the heart in the human subject it has occurred to me to witness.[1] Besides, were the organ sensitive to any notable degree, it seems at the least strongly probable that violent palpitation would, under all circumstances, be more or less seriously painful. Yet we know, as a clinical fact, that the most violent jogging motion of the heart may, and does, take place in some persons without the least feeling of genuine pain.

318. Now whether with or without structural change the heart ever becomes the seat of morbid tactile hyperæsthesia, may be considered unproven. It is unusual, to say the least, in cases of endocarditis, to find pressure in the præcordial spaces painful, unless pericarditis be superadded. And the very positive tenderness under pressure noticeable in many cases of the latter inflammation, must probably be referred to the serous membrane itself. Patients with dilated heart sometimes shrink from pressure, especially if abrupt, on the corresponding part of the chest wall; I have known similar sensitiveness in cases of diseased aortic valves. On the whole, it seems reasonable enough to admit the probability of the condition, though the fact be hardly susceptible of satisfactory proof.

(*c.*)—SPECIAL HYPERÆSTHESIA.

319. Without attempting to dispossess the Hallerian doctrine of inherent irritability, as a cause of muscular action, and conceding the motor power of the nerve-ganglia of the heart and of their

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[1] U. C. H., Males—case published by Dr. Boswell Reid.

rhythmic centres, still we cannot reasonably doubt that the degree of sensitiveness of the nervous expansion on the endocardial surface to the stimulus of the blood, must play a part in regulating the activity and energy of the systolic movements. There must be a normal standard of that sensitiveness, just as there is in the case of the pulmonary nerves in regard of the stimulus of atmospheric air.

320. If, then, there exist such a normal standard, increase above or decrease below par are conceivable states of disturbed dynamism. Are they observable ones? I have no certain evidence pro or con. But it has appeared to me sufficiently likely that some examples of palpitation, for which no one of the acknowledged causes can be discovered, may really be engendered through such special hyperæsthesia. Just as abnormally irritating blood is an admitted cause of undue excitement of the endocardial surface, and through this of palpitation, the nerves being healthy—so, conversely, blood of naturally stimulant quality may prove the indirect cause of overaction through contact with an over-sensitive rete of nerves.

II.—CARDIAC PARÆSTHESIÆ.

321. Anomalous sensations, or paræsthesiæ, of various kinds may be experienced within and about the area occupied by the heart. The usual difficulty is exhibited by patients in their attempt to describe the precise nature of these sensations. But in some instances they seem to correspond more or less closely to the formication, heat or coldness, numbness, tingling, &c., well-known in the limbs; sometimes, special to the organ, they are spoken of as a sense of rolling, bubbling within, twisting of, or sudden falling downwards or backwards of its mass.

322. There is an anatomical reason for connecting these sensations with the cardiac nerves: they occupy precisely the same sites as the pain of simple cardiac neuralgia, or that of fully developed angina. There is besides a physiological justification for connecting the two things; these paræsthesiæ occasionally produce leipothymia, or actual syncope, palpitation, fluttering action of the heart, or irregularity of pulse without palpitation. They are sometimes, even in the male, accompanied with globus and sense of choking.

323. Such anomalous sensations are often made light of, because they do not amount to actual pain. Yet they are probably quite as significant, as really acute suffering, of an unsatisfactory and more or less dangerous condition of cardiac function. I am led to apprehend from cases which have fallen under my observation in private practice (they are rarely to be seen in hospitals), that where they unmistakably exist, these paræsthesiæ indicate a want of perfect textural soundness in the heart. Nothing may be physically wrong—auscultation gives apparently normal results: yet at any moment the mechanics of the organ may be dangerously perverted.

324. Cardiac paræsthesia, sometimes connected with passing con-

gestion either of the cavities of the heart, or possibly of its actual tissue, is occasionally linked to organic disease, either of the muscular substance, or more commonly of the valves. But in the idiopathic form it seems a reflex neurosis, in which irritation of the pneumogastric forms the first link in the chain; even here, as just suggested, the probability is, some unhealthy condition of texture, too delicate for clinical discovery, affects the organ—otherwise it seems difficult to understand why cardiac paræsthesiæ should not be materially more frequent even than they actually are. Agents which seriously affect the inhibiting function of the vagus in regard of the heart's movements—for instance, digitalis and digitaline—when exhibited in poisonous doses, produce various cardiac paræsthesiæ, sometimes actually acute pain.

III.—CARDIAC ANÆSTHESIA.

325. Of anæsthesia, or sensory paralysis, of the heart, little is known. Nothing has, indeed, as far as I am aware, been clinically ascertained concerning annulled tactile sensibility of the organ. On the other hand, impairment of its specific sensory innervation is so mixed up, nay, identified with, certain forms of failure of its motor power, that the subject may be most conveniently taken up in association with that of cardiac motor paralysis [326] and syncope [398].

B.—MORBID MOTOR STATES OF THE HEART.

I.—CARDIAC MOTOR PARALYSIS.

(*a.*)—FROM DEFICIENT INNERVATION.

326. Motor paralysis of the heart, dependent on failure of innervation, is produced in four essential ways.

327. (*a.*) It may be centric: as where intense emotion, either painful or pleasurable, or sudden and general injury of, or shock to, the encephalon or cord, causes instantaneous cessation of the heart's contractions.[1]

328. (*b.*) It may be reflex through the ganglionic system; as where a blow on the epigastrium causes fatal syncope.[2] It may be reflex, also, through the spinal nerves, as where severe injury to an extremity, or to an extensive islet of skin, produces fatal depression of the heart's action.

329. (*c.*) Cardiac paralysis may also be induced through direct

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[1] That the heart continues to beat after the gradual removal of the brain and spinal cord in animals does not shake the truth of this statement of facts, practically well known. Besides, it is certain that if the brain or cord be suddenly crushed in an animal, the heart ceases to beat.

[2] Marshall Hall proved that sudden stoppage of the heart's action might be caused by "violently crushing the stomach with a hammer," even where the communication through the cord and encephalon was impossible—those centres having been previously removed or destroyed (in an eel). Dis. of the Nervous System, 1841, p. 128.

interference with the supplying portions of the ganglionic system. At least, cases now and then occur in which the pressure of enlarged bronchial glands, mediastinal tumor, or aortic sacculated aneurism on the cardiac nerves, has caused occasional interruption of contraction. In a very remarkable instance of suspension of action for five or six beats at a time, which ended fatally, the explanation was found in a knotty, diseased, and compressed state of the great cardiac, with the vagus, and the phrenic nerves.[1]

330. (*d.*) The continuance of cardiac action after cautious and gradual piecemeal removal of the brain and cord, and the vitality of monstrous fœtuses, born without either of those parts of the nervous system, point to the non-dependence of the heart's activity upon them, and to the reality of intrinsic production of nervous force as the main source of movement. Remak's discovery of ganglia on the nervous filaments within the texture of the heart, seems to give the impress of positiveness to this view concerning the heart's motility.

Now, it is infinitely probable, changes occurring in the ganglia themselves, either dynamic or structural, may be the immediate cause of suspended pulsation of the heart in some cases: but the clinical proof is yet wanting. Still in certain fatal cases of chloroform-inhalation, where death occurs through the heart, it seems reasonable to believe the vapor, acting directly on the cardiac ganglia, plays a part in instantaneously arresting their activity [397].

331. The practical relationships of this subject are considered under the head of SYNCOPE.

(*b.*)—FROM EXHAUSTED CONTRACTILITY.

332. That deficient action of the heart depends sometimes on purely dynamic failure of the specific vital property of its sarcous element, contractility, the nervous supply remaining unchanged in quality and amount, seems hardly susceptible of proof. To demonstrate in any case of failing contraction, that the stimulus is positively unaltered, but the faculty of responding to that stimulus destroyed, exceeds the clinical power of the present day.

Nevertheless, analogy suggests the possibility of such a state. The sudden cessation of contractile action of the bladder in cases of stricture of the urethra, where immense force having been used to effect the discharge of perhaps not a quarter of its contents, any further effectual strain of its walls becomes impossible, most probably depends on temporary loss of contractility. The sufferer is at least conscious of vainly directing the full force of volition to excite the viscus to further contraction.

Besides, it would seem to follow, from the experiments of Dr. Blake, that certain agents injected into the blood really do arrest cardiac action, not by perverting its innervation, but by annulling its muscular irritability [394].

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[1] Heine, Müller's Archiv., 1841—quoted by Romberg, Dis. of Nerv. Syst., vol. ii.

333. No reference is of course intended here to cases in which statical change affects the sarcous structure. The minutest amount of such change confessedly and certainly nullifies in the most complete manner any amount of activity of motor innervation in the spots actually diseased.

II.—Cardiac Spasm.

334. Does the heart ever become the subject of true spasm, either clonic or tonic? A positive answer, founded on clinical evidence, cannot in all probability be given to this question.

335. But Ed. Weber has shown that portions of the heart may be brought into a state of persistent tonic contraction, and cut off from the rhythmical movements of the rest of the organ, by the interrupted galvanic current and by the direct application of strychnia to the endocardial surface. Hence there is a possibility, at the least, that perverted motor innervation may cause more or less enduring spasmodic contraction, local or general, of the heart.

336. Besides, it is not very uncommon to find the left ventricle so tightly contracted, on post-mortem examination of persons dying from various diseases, that it requires an effort with the fingers to open out the mitral orifice. I must confess ignorance as to the precise conditions preceding death in such cases; but the observations of M. Cruveilhier on simulated concentric hypertrophy of the heart throw some light on the matter. It is certain, at the least, that in cases of death from tetanus, knotty spasmodic contraction of the organ has sometimes been observed. Some further notice of this subject will be found with the description of Angina Pectoris.

III.—Cardiac Cramp.

337. Cramp of voluntary muscle clinically consists of local tonic contractions, plus more or less agonizing pain. It is commonly believed the pain specially depends on dislocation or rupture of muscular fibres, or possibly even of nervous filaments. Protrusion of the primitive fasciculi through ruptures in their containing sheaths has been microscopically proved to occur in the voluntary muscles in cases of tetanus; but I am not aware that structural injury to nerve has been similarly demonstrated.

Such mischief in the sarcous structure of the heart remains, if real, to be detected. The phenomena of certain fatal anginal seizures, make it, however, sufficiently probable that the search would not always be vain. Patients occasionally, and of their own accord, liken the pain of angina to that of external muscular cramp.

IV.—Perversion of Rhythm and of Force of Cardiac Contraction.

(*a.*)—OF THE ORGAN AS A WHOLE.

838. There are numerous affections of the heart of a statical kind which produce irregularity in the time and force of systolic action—such as sudden rupture of a chorda tendinea, or of the substance of a valve, accumulation of coagula in the interior of the cavities, etc.; the irregularity in such instances becomes more or less readily intelligible on mechanical principles. But in a very large proportion of cases of irregular rhythm, there is no evidence of mechanical difficulty in the organ; we are obliged to admit in such cases either that mechanical mischief, beyond existing powers of clinical detection, exists, or that the disturbed rhythm is purely dynamic. The latter notion is in general more probably the correct one—and to this form of perversion alone we propose now to direct attention.

839. It may be assumed that modified nervous influence is the intimate cause of dynamic abnormal rhythm. And that the modification may be centric or reflex seems amply shown by the clinical conditions under which irregular action is observed. Nor can there be any doubt, if we trust the results of recent physiological inquiries, that the intrinsic ganglionic system of the heart may sometimes be independently at fault; though of this clinical demonstration is neither at present forthcoming nor likely soon to appear. Thus, Volkmann's success in locally disturbing the rhythm of the organ by cutting and chopping its texture in various spots and manners, while it seems to show pretty conclusively that rhythmical action is under the immediate influence of local powers, limited in each instance to certain fixed areas, affords no clue to the comprehension of irregularity of beat occurring in the human subject. Nay, more, though we accept the existence of "rhythmic nerve-centres" as positively demonstrated, and admit that these centres in the nerve-ganglia, by discharge of nerve-force at regular intervals, induce the fixed rhythmical contractions of the organ (mere motor power depending on quite a different agency[1]), we shall have long to wait before clinical demonstration of the part played directly and exclusively by mishap, dynamic or textural, to these rhythmic centres, can become a possible achievement. The problem of irregular rhythm is, in truth, one of infinite complexity. Thus, the readiness with which time-regulated action is perverted is shown by an experiment of E. H. Weber, proving that even slight changes of temperature gravely modify the frequency of the heart's beat. How many other conditions must there not be, of like or greater

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[1] Pathology long since proved this by exhibiting force changed, while rhythm remained natural; or, *vice versa*, irregularity of beat with power of contraction sustained at par.

potency, the influence of which the observer of the present day scarce even suspects![1]

The reflex influence of the gastric branches of the pneumogastric seems clearly established clinically. The passing irregularity of action, which follows in some persons the mere ingestion of a full meal, and the graver and more continued disturbance, occasionally attending different forms of dyspepsia, seem otherwise inexplicable. And, again, the perversion of rhythm immediately induced in some exceptional persons by certain articles of diet, as green tea, is probably a reflex effect of specific influence on the vagus nerve. Nor is the agency of the pulmonary branches less certain. A remarkable illustration, occurring in his own person, has been recorded by Dr. Nooth: a shot had lodged in a bronchial tube; subject to asthmatic seizures and extremely irregular pulse, whilst the foreign body remained in its place, the patient lost both symptoms on its expectoration.[2]

339*. The different varieties of irregular beat have already been mentioned [78, 172]. A simple intermittence appears to signify *pro tempore* cessation of power to contract, and may consequently be looked on as a *perpetually recurring, short-lived syncope;* in support of this view stands the fact that some persons, subject to intermittent action, are conscious of instantaneously transitory faint feelings at the moment of its occurrence.

340. Although, as a rule, irregularity of cardiac rhythm must be looked on *per se* as of unfavorable import, it is indubitable that persons are occasionally encountered whose health is excellent, though their heart's action is more or less constantly irregular. The constancy of irregularity is subject to great variety—inexplicably so for the most part. Individuals whose heart's beat is habitually irregular in ordinary health may have perfectly rhythmical action during pyrexia, either as a constant or an occasional state, so long as the febrile excitement endures. Subjective consciousness of irregularity, either in the form just referred to, or in any other shape, is by no means invariable, and is more common where palpitation is present than where this is absent. There are certain idiosyncrasies mixed up with this perversion of very singular character: the case referred to above, where a very small quantity of green tea will, in persons whose ordinary beat is rhythmical, immediately induce irregularity, is a sufficient illustration.

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[1] Many years ago Dr. Neil Arnott, in his "Elements of Physics," suggested that the rhythmical movements of the heart might be referred to accumulations of force, discharged at regular intervals, likening those movements to "some electrical phenomena in which there are successive accumulations and exhaustions of power." ("Elements of Physic," fifth edition, 1833, vol. i. p. 597.)

Fresh interest has been recently given to the subject of rhythmic action by the lucid exposition of Mr. Paget, who, in a series of propositions of increasingly wide generalization, finally endeavors to establish time-regulated rhythmic motion as not only a dependence upon, but a necessity of, *rhythmic nutrition*. (Croonian Lecture, Royal Society, May, 1857.)

[2] Trans. Soc. Impr. Med. Knowledge, vol. iii. p. 5.

(b.)—OF DIFFERENT PORTIONS OF THE HEART IN RESPECT OF EACH OTHER.

341. The natural synchronism of action in the two ventricles, in the aorta and pulmonary artery, and probably, though less demonstrably, in the auricles, may be variously perverted.

Of these perversions of action, which are solely revealed by auscultation, the history, as far as I have made it out, has been given under the head of Reduplication of the Heart's Sounds [176].

V.—Action Perverted in Frequency.

(a.) INFREQUENT ACTION.

342. It is conceivable the heart shall contract less frequently than natural, either because (1) normal innervation is in some way perverted; or because (2) nervous influence continuing normal, the contractility of the heart's fibre and its faculty of responding to stimulus are impaired. Facts illustrating both forms of infrequent action, to all degrees between the normal rate and one of twenty-four per minute are clinically known.

Whatever be its particular mechanism, an infrequent rate of beat may be attended with quick and abrupt or with slow and drawling ventricular contraction; and the pulse may, or may not, accurately reproduce the number and the qualities of the ventricular action.

343. (1.) The tendency of concussion of the brain, of encephalitis in some cases, and of a variety of other cerebral affections, to slacken the rate of cardiac action, has long been practically recognized. Definite notions on the mode of connection between the cause and its effect have been wanting: it has simply been presumed that by shock of, or altered circulation in, the nervous centre, the *exciting* force of innervation sent to the heart has been *diminished*.[1] Recent experiments by Edward Weber, Pflüger, Lister, and others, have, however, been held to prove the converse mode of influence—that, in fact, *increased* activity of the *inhibiting* functions of certain portions of the encephalon (*corpora quadrigemina*), of the medulla oblongata and of the vagi nerves is produced by the cerebral conditions enumerated, and through that increase the heart's rate of movement slackened. The mode of influence is supposed to be the same as that observed from galvanic excitement. The experiments of Traube on digitalis seem to support this view of the mechanism of slackened action. Having first ascertained

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[1] The majority of observers localize this paralytic condition in the encephalon itself; and I think with reason. Others, however, think the deficient energy traceable to the vagi. Thus, Zengerke, finding that the pulse invariably slackened when the brain of the dog was pressed on with the finger (through openings made in the skull), refers the effect, not to the direct pressure on the brain itself, but to that indirectly made to bear on the vagi nerves at the base. But he gives no conclusive reasons for the adoption of this view. (Ranking's "Retrospect," vol. xxvi.)

that the pulse of a good-sized dog fell in sixty seconds from 132 to 24 beats per minute under the influence of eight grains of extract of digitalis, injected in solution into the jugular vein towards the heart, and that the frequency rose again in the course of about twenty minutes under the influence of four other injections, alike in amount, to 174 beats per minute, Traube proceeded to test the agency of the vagi in the matter by two separate series of experiments, in one of which the vagi were divided before, in the other after, the administration of the digitalis. Now the results appear to warrant his inference, that infrequency of pulse is caused by stimulation of the inhibiting function of the vagus, frequency by paralysis of that inhibiting function. Whether the slackening of the heart's motion, which ranks among the effects of lead-poisoning,[1] and of aconite, is similarly produced, remains to be determined.

344. (2.) Inobedience to ordinary stimulus, and also incomplete deficient readiness to contract, on the part of the heart, are shown in the infrequent action attending fatty metamorphosis of its fibre. But even in this case the nervous dynamism of the organ also is at fault; for no direct ratio holds between the amount of sarcous destruction and the slackness of the pulse. Mere weakness of the walls of the ventricles will not retard the frequency of their beat: in attenuated dilatation the pulse may be frequent; in the soft, flaccid, and weak states of the heart, occurring in typhus and typhoid, the action is invariably frequent.

345. Infrequent action of cerebro-spinal origin is strong; of cardiac origin weak: in either variety the heart may not beat more than from twenty to forty times in the minute.

346. There are other circumstances under which slackness of pulsation must probably be referred to the double cause. The peculiarly infrequent pulse of some persons, whose health is in all respects excellent, belongs to the number. So also does the slackened and enfeebled action that in certain cases marks the active period, or the convalescent stage, of acute diseases, attended with debility and nervous exhaustion; I have seen this well defined in the acute stages of bronchitis, and during recovery from diphtheria, for instance: but, doubtless, the retarded beat is not specifically an attendant on the former of those diseases rather than on any other of a weakening character—probably, however, it possesses specific significance in the latter.

In cases where infrequent rate of beat is a physiological, and probably congenital peculiarity,[2] unattended with cardiac, respiratory, or cerebral disturbance, febrile diseases increase the frequency and strength of the action, and lessen any existent tendency to irregularity of rhythm.

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[1] In 678 of 1217 cases of lead-colic analyzed by M. Tanquerel des Planches, the pulse ranged between 30 and 60.

[2] Vide an interesting case by Dr. Macdonnell, in "Brit. Amer. Journal of Medicine."

(*b.*) OVER-FREQUENT ACTION.

347. The mechanism of increased frequency of the heart's pulsation, one of the most familiar phenomena of disease, is far from being fully understood. Beyond question the same rationale is not applicable in all cases.

348. Let the frequent pulse of pyrexia be taken by way of illustration. Upon which of the co-essential phenomena of febrile action does the accelerated beat depend? On the abnormal condition of the nervous system, which, if the teachings of Chomel be correct, forms the principal element of fever—involving a plus state of innervation of the cardiac plexus, or of the cardiac ganglia? On the rise in temperature of the solids and fluids—a notion supported by the relatively exciting and depressing influences of atmospheric heat and cold? On the irritating influence of the blood—altered as this is in composition, both in the proportions of its normal elements, and by the accumulation of abnormal constituents, through defective excretion, excessive disintegration of tissue, absorption of morbific material from without, and septic changes within itself? To which of these influences alone, or in combination with others of the group, the accelerated action is immediately referable, it is impossible positively to affirm.

Nor is our ignorance less of the precise mechanism, whereby spanæmia hurries the heart's action—though changed blood, disordered innervation, and accelerated rate of breathing, all probably play their several parts.

VI.—Action Perverted in Quickness.

349. Each independent beat of the ventricles may be effected with abruptness and quickness, or with deliberation and slowness. One or other of these conditions may exist while the rate of successive beats is either frequent or infrequent. Both varieties are known clinically. The lagging, sluggish contraction of *some* hearts affected with hypertrophy is significant enough in diagnosis, when well defined.

350. Infrequent action, as already seen, attends a certain share of cases of fatty change of the heart: each action is then quick. Each contraction, long in coming, is brief in duration; the pulse is infrequent and quick.

351. In certain cerebral affections the ventricular action, lessened in frequency, is besides slackened in speed: the pulse is infrequent and slow.

352. Again, in many cases of non-rhythmical palpitation, the varying amount of quickness or slowness in the ventricular action adds an important element to the mass of irregularity.

353. The specific mechanism of this imperfection, where purely dynamic, is unknown; its relationship to fatty atrophy is glanced at with the history of that disease.

VII.—CARDIAC TREMOR.

854. True tremor appears from Volkmann's inquiries to depend on weakened tonicity of muscle; this tonicity in turn to be sustained by impulses from the spinal cord—impulses successive, it is true, but so closely successive that the effect of any one has not had time to die away before its successor has sprung into existence. Thus is explained a *constant* effect from an *interrupted* action. But the effect, like the action, becomes perceptibly non-continuous, if the impulses slacken in frequency. The result is tremor.

855. Now the existence of such tremor in the heart is even more problematical than that of any of the perversions of muscular action yet mentioned. Still the difficulty of accounting for the occasionally excessive irregularity both in *force* and rhythm of the heart's action, justifies us in not at once rejecting any plausible hypothesis. It may be that certain forms of irregular fluttering palpitation in part consist of genuine tremor. That is, in addition to irregular movement arising out of mechanical difficulties and modified innervation of the heart's *contractility*, impairment and unsteadiness of the nervous force presiding over its *tonicity*, may by possibility contribute a share of feeble, wavering, non-rhythmical motion.

856. Were this view sustained, the experiments of Volkmann (which prove that tremor of a voluntary muscle may be imitated by slackening the turns of the weak rotatory galvanic apparatus, so that the impulses may succeed each other at appreciable intervals of time) suggest by inversion a mode of treatment of fluttering palpitation.

§ II.—COMPOSITE DYNAMIC CHANGES, OR FUNCTIONAL DISEASES.

357. THE clinical conditions, known as Functional Diseases of the Heart, are worked out of the elementary disturbances of cardiac innervation and motion, just reviewed, variously and capriciously associated. The list comprises simple palpitation or increased action; irregular action, without or with palpitation; decreased action and syncope; infrequent action; slow action; angina pectoris; and pseudo-angina pectoris.

I.—SIMPLE PALPITATION.

358. By simple palpitation is meant increase in the force of the heart's contractions, unattended with any change of rhythm, and not necessarily with increased frequency of beat.

359. The amount of force may be slightly above the par of health; or sufficient to shake the chest or entire body of the patient, or even the bed on which he lies.

360. *Symptoms.*—Consciousness on the part of the patient of the heart's action seems, at the least, an almost invariable attendant on palpitation. A variety of subjective sensations may be enumerated, occurring singly, in groups, or all of them combined. Such are choking sensations; a form of *globus*, or feel as if the heart were "jumping into the throat," and the eyes bursting from the sockets; præcordial anxiety; faintness, actual syncope, or partial unconsciousness. If præcordial pain exists, it commonly amounts to little more than a sense of dull, aching soreness; but I have in a very few cases known very severe pangs, similar to those of angina, attend the paroxysm: such pangs are sometimes relievable by pressure of the hand, even where the patient cannot endure the recumbent posture on the left side.

361. Hurry of respiration, to any very notable amount, is of rare occurrence; it does sometimes, however, reach a degree out of proportion with the pulse, giving the patient the appearance of a person out of breath with running. Tinnitus aurium; vertigo and confused vision; cephalalgia, heat of head and flushed face; and clammy coldness of the extremities, add themselves to the list of disturbances.

362. Although this be more common, when rhythm is perverted, yet even in attacks of simple palpitation there may be extreme

general distress and fear of death—singularly enough, more of this, often, than in cases of palpitation of organic origin: the consciousness of local suffering is rather in the ratio of the general and local sensibility, than of actual palpitation.

363. In cases of long duration the eyeballs seem to enlarge, and distinctly protrude to an unnatural amount from the orbits. I once saw a case where this enlargement gave more annoyance and apprehension to the sufferer than all the other evils of the complaint combined.

364. Overgrowth of the thyroid gland proves an occasional, but as far as I have seen, very rare effect of long-continued palpitation.

365. *Physical signs.*—The impulse is too extensively visible, but the apex-beat natural in site. If the heart be a well-nourished one, the impression it gives to the hand laid on the cardiac region, is that of a *blow*—the impulse may even be somewhat heaving; if a feeble organ, the impression is that of a *slap*. Systolic basic thrill may, under special circumstances, be felt [85]. Habitually, the area of dulness remains unaltered, but extension to the right of the sternum may occur, in prolonged paroxysms especially; dulness is never carried upwards. The first sound is too loud and clear at the mitral apex, and somewhat abrupt and short; the second is lower-pitched and less clicking than natural at the mid-sternal base. The first sound may be loud enough to be audible, both to the patient and to bystanders, at a distance of some inches from the chest. Reduplication of the second sound at the base is common; and a distinct metallic ring, or a pericardial rub, may accompany the shock at the mitral apex.

366. Will simple palpitation produce murmur in a structurally healthy heart? If there be the slightest coexistent spanæmia (so slight that in the state of ordinary action the sounds are perfectly normal), basic systolic murmur will most certainly be generated. But murmur of the same seat and time may also occur, both in males and females, during violent palpitation, where all attainable evidence points to the non-existence of any morbid state of the blood [204]. I suspect that mere palpitation may also cause passing mitral regurgitant murmur of dynamic mechanism [205]; but of this I am not sure.[1]

367. The aorta, the carotids, and the arteries generally, beat with undue force, sometimes exceeding that of the heart itself—more especially in spanæmic persons.

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[1] In the valuable work of Dr. Stokes, an opinion is by inference ascribed to me on the subject of inorganic cardiac murmurs, differing from that I have really professed. Dr. Stokes seems to infer that I deny the possible production of systolic murmur at the apex, independently of textural disease of the mitral orifice (Diseases of the Heart, p. 496). Far from this, I have always strongly insisted on the probability that *dynamic* murmur of this site and rhythm really occurs; but cardiac murmur of *blood-origin* I have supposed, and still suppose, to be always basic in seat. I have never yet heard in a purely chlorotic woman a murmur having all the characters of a mitral regurgitant one.

368. The radial pulse is quick, sudden, forcible (yet without real strength, for it yields easily to pressure), and over-frequent, when palpitation occurs in nervous, anæmic people. In the vigorous and plethoric, although the heart be perfectly free from over-nourishment, the beat may be full, hammering, heavy, and resisting: its frequency normal, or even below par.

Very slight jugular pulsation may, I think, occur independently of demonstrable tricuspid regurgitation, or of hypertrophous dilatation of the right ventricle.

369. *Duration.*—The heart of certain sufferers from palpitation is constantly in a state of more or less marked over-action: they are never perfectly free. But such cases are, on the whole, exceptional; the complaint is essentially paroxysmal. Lasting for a few minutes, an hour, or with remissions for days together, a fit of palpitation frequently terminates by sleep. In hysterical women a copious discharge of watery urine may occur at the time of release, but it is by no means a constant event.

The time, during which a patient remains subject to attacks, varies infinitely; as likewise the length of the intervening periods of freedom.

370. *Causes.*—Early adult age, the female sex, and the nervous temperament are commonly held to predispose to palpitation. I do not deny the inflence of any of these conditions; but the gravest examples of palpitation I have met with have certainly occurred in males.

371. The exciting causes range themselves under the heads of perverted innervation, morbid conditions of the blood, diseases of the lungs, and mechanical influences.

372. Perverted innervation may be centric, reflex, or intrinsic. The *centric* mechanism is exemplified by palpitation from emotion of various kinds, over-study, mental excitement, the abuse of alcoholic liquors and tobacco. To this category belongs palpitation attending certain centric nervous diseases, as hysteria, epilepsy, spinal irritation, chorea, hypochondriasis, and cerebral erethism with insomania. To *reflex* mechanism may be traced over-action excited by dyspepsia of all kinds; by certain articles of diet, as strong tea; liver disease; intestinal irritation, verminous or other; ovarian irritation and disordered menstruation; venereal excesses; masturbation and spermatorrhœa; and intercostal neuralgia. The play of the *intrinsic* ganglionic system of nerves is difficult of proof; but it is probably not the less real. It has already been referred to [339].

373. Blood altered in composition, it is to be presumed, irritates the nerves presiding over the specific excitability of the heart. If so, the mechanism becomes really reflex. Be this as it will, spanæmia, plethora, uræmia, hyperinosis, hypinosis, gouty and rheumatic blood, seem all capable of inducing palpitation more or less actively.

374. The influence of *pulmonary disease* is less frequently apparent than might be supposed. Still, the over-action, entailed by chronic bronchitis and emphysema on the right side of the organ, extends to the left, and constitutes a form of palpitation.

375. To the *mechanical* order belong cases referable to distortion of the chest, pressure from abdominal fluid or tumors, pregnancy, tight-lacing, sideward displacement of the heart by pleuritic effusion, &c.

376. *Treatment.*—The treatment embraces (*a*) the management of the fit; and (*b*), during the intervals of seizures, the removal of the conditions exciting them.

377. (*a*.) It may be possible sometimes to remove the cause of a fit at the time of its occurrence, as when palpitation depends on acid and flatulent dyspepsia. But in the majority of cases during its actual presence our exertions are directed rather towards lessening the intensity and shortening the duration, than removing the cause of the seizure.

Thus the duration and amount of suffering may be diminished by free doses of antispasmodics, assafœtida, musk, and valerian, especially in hysterical persons; by diffusible stimulants, ammonia, the æthers, and very strong coffee; by narcotics and sedatives, opium, hyoscyamus, hydrocyanic acid: if the excitement of the organ be great, and its action not distinctly irregular, digitalis may be used with propriety. Acidity and flatulence, frequent causes of palpitation, may be corrected by soda and cajeput oil; a loaded stomach freed by an emetic. If plethora of the sthenic kind be present, cautious venesection is advisable: if of the asthenic, digitalis tranquillizes the organ speedily. Should a gouty or rheumatic state be discoverable, colchicum, guaiacum, and ammonia, and irritant stimulants to the joints, are the best remedies. Heat may be applied to the extremities; but the application of ice over the heart is a dangerous practice, especially if the rhythm of the organ be in the smallest degree affected.

378. (*b*.) The measures to be adopted in the intervals will often vary in their essential feature with the nature of the constitutional or local fault that excites the paroxysms. But there are besides some general rules, applicable under all circumstances, which may be set down here. Persons subject to palpitation should avoid stimulants in any quantity, over-exercise, over-sleep, emotional and intellectual excitement. Hydrocyanic acid, aconite, digitalis, and belladonna, internally, varied according to circumstances (the latter also in the form of plaster over the heart), coupled with the use of the shower-bath, strict attention to diet, regularity of bowels, cheerful occupation, and, lastly, change of air, will either remove the tendency to palpitation altogether, or greatly mitigate the severity of the seizures. Should there be the least evidence of spanæmia, iron is indispensable.

II.—PALPITATION OF IRREGULAR RHYTHM AND FORCE.

379. This variety of palpitation is in its extreme forms essentially paroxysmal: persistency of its highest degree and continuation of life would be, in point of fact, incompatible.

380. *Symptoms.*—A typical paroxysm is characterized as follows. *Motorially:* the action of the ventricles is remittent, hesitating, or anticipating; intermittent at regular or irregular intervals; the force of contraction now increased, now diminished; the sequence of the auricular and ventricular contractions irregular; the synchronism of both ventricular, as also of both auricular, contractions lost; the valvular and papillary actions more or less perverted in rhythm; the rate of motion perpetually changing; and associated with all this, probably, in some cases, genuine tremor of the organ [354]. *Sensorially:* combinations in all conceivable varieties of cardiac paræsthesiæ and hyperæsthesiæ—making up in their totality various amounts of so-called "præcordial anxiety."

381. A sort of order in disorder is sometimes observed—a regular recurrence of certain conditions of irregularity taking place at fixed intervals; in these rare cases cycles of irregular action really exist.

The radial pulse of course shares in the rhythmical disturbance. The precise relationship is, however, very difficult to trace; the weaker class of systoles fail constantly to impress the artery at the wrist. This matter has, however, been already considered [173]. The rate of pulsation at the wrist may, in one and the same case, be *momentarily* marked at fifty in the minute, the next instant rising to a hundred and fifty, or becoming uncountable from combined frequency, irregular rhythm, and unequal force.

382. Syncopal tendency, or actual passing syncope, pallor of face, alternating it may be with lividity, breathlessness, thirst, nausea or vomiting, disposition to relaxation of the bowels, haggard and anxious countenance, intense general uneasiness, jactitation, alarm, and even fear of death, and clammy perspiration, mark at once the reflex suffering of allied organs, and the deep sympathy of the frame at large with the disordered function of the heart.

383. Immediately on the close of an attack, copious watery flux from the kidneys sometimes occurs.

384. *Physical signs.*—The impulse may be slight in amount, fluttering in character; or irregularly forcible, now strong, now lost, both to the eye and hand; in extreme cases the motion becomes jogging, trembling, rolling, quivering, or indescribable.

A heart beating, now fifty, now one hundred and eighty or more times in a minute, now with excessive force, the next moment with such feebleness that the hand scarcely catches the impulse, cannot fail to become more or less clogged and obstructed internally. The evidence of this appears not only in the syncopal and suffocative tendencies of such fits, when at all prolonged, but in the increased area of the heart's dulness—especially to the right of the sternum.

The rhythm of the sounds is, of course, altered—reduplication is common [176]. Their strength and quality vary from moment to moment—the general tendency of the first of the two, especially, is to shortness and undue sharpness of tone; but occasional forcible contractions may give a healthy systolic sound, masked possibly by parietal shock or pericardial rub. On the occasional occurrence of dynamic murmur I have already dwelt sufficiently.

385. *Causes.*—The extrinsic causes of palpitation of this form are the same as those of the simple variety. The intimate mechanism of irregularity has already been considered [338]; the intrinsic conditions in the heart and its appendages which bring that mechanism into play—which cause a given heart to palpitate irregularly rather than regularly—are not only beyond our ken, but defy our means of investigation.

386. *Prognosis.*—The prognosis is, absolutely speaking, serious, and relatively much more so than in the previous variety. Mere irregularity of rhythm, if extreme, cannot be regarded as a matter of light importance—notwithstanding exceptional individuals exist, who, by some peculiar idiosyncrasy, enjoy good health, though their pulse be more or less irregular [340]. But in these persons the organ, though irregular in its beat, is normal in force of action—now the case becomes a very different one where palpitating motion is added to irregularity. And though by possibility the combination may exist without structural change, I confess I have rarely encountered it, where there was not either certainty or strong suspicion of alteration in the heart's texture. At all events, I am persuaded that when elderly persons are the frequent subjects of what is called "stomach palpitation," the assumption that, because no valvular signs or evidences of marked hypertrophy or dilatation are discoverable, therefore the heart is sound, is a positive error. There may exist (as demonstrable post mortem) soft flabbiness of texture or incipient fatty atrophy, which clinically gives no appreciable sign. Death may, under such circumstances, actually occur during the fit.

387. *Treatment.*—In treating a paroxysm of this, as of the previous, form of palpitation, the search after causes must never be omitted: that search is even more important as a guide to management in the intervals.

388. Stimulant and antispasmodic medicines are especially indicated in this form; if there be local pain, opium must be had recourse to. Mustard poultices to the præcordial surface seem, in some cases, to help in regulating the heart's action; they lessen local distress, and never do mischief. Digitalis is a remedy of doubtful propriety, even where the rate of motion is much accelerated; it may readily contribute to coagulation of blood within the cavities.

389. In the intervals, iron and other metallic tonics, the use of

the shower-bath, and an invigorating regimen generally are indicated. When this can be effected, change to a climate at once mild and bracing should be tried. Walking exercise under favorable climatic conditions sometimes proves very serviceable, where no structural change has occurred in the heart: I have known a pedestrian excursion in a mountain district, under such circumstances, prove singularly restorative.

III.—DECREASED ACTION AND SYNCOPE.

390. Decreased action of the heart, affecting force and frequency of contraction, if carried to extremes, produces syncope. In its minor degrees, and if an habitual state, it entails general languor of the functions. Let us first consider the phenomena in their extreme degree.

A.—Syncope.

391. Syncope, in its most perfect condition, is a state of apparent lifelessness, in which failure of the heart's propulsive action forms the first link in the chain of events tending to somatic death. The second link, suspension of the functions of the encephalon, is followed, almost accompanied, by the third, cessation of breathing.

392. *Causes.*—The conditions which appear to *predispose* to syncope are the female sex, early adult age, nervous and especially hysterical temperament, spanæmia, general weakness, and idiosyncrasy. Cases every now and then occur in which the importance of the latter condition appears paramount. I have met with some half dozen persons in the course of my experience who, perfectly sound in all their organs, were in the habit of fainting, sometimes without the intervention of any traceable exciting cause, sometimes under an influence so slight that in the mass, even of feeble and nervous women, it would have proved wholly incapable of arresting the actions of the heart. Doubtless in such persons there must be some inherent weakness or specific impressionability in the cardiac plexus, the cardiac ganglia, or the connected centric portion of the nervous system.

393. The *exciting* causes of syncope, excessively numerous and diversified, may be arranged under three heads, *dynamic*, *hæmic*, and *organic* including *mechanical.*

394. *Dynamic.*—In this group of cases the proximate cause of stoppage is either failure of the special irritability of the heart, paralysis of its motor nerves, or spasm of its fibre: and in the majority of cases it is impossible to affirm with positiveness which has been the mechanism at work.

But in this group, without attempting their finer distinction, there can be no error in placing syncope from the paroxysm of angina; from fatty heart, without rupture or intracardiac coagulation; from the influence of certain drugs and chemicals, as digitalis [343], aconite, tobacco,[1] antimony, hydrocyanic acid, colchicum, oxalic acid, and arsenic;[2] from the effects of lightning; from excessive galvanic stimulus [407]; from prolonged stay in the warm bath. Here, too, might be placed the fatal syncope which sometimes puts a close to the lingering career of phthisical patients, and which, it is suggested by M. Louis, may depend on "fatigue of the heart."

Direct injury to the brain, as in general and severe concussion,[3] sudden mechanical impression on the semi-lunar ganglia, and solar plexus, as from a blow in the epigastrium; specific impressions on the nerves of sense, as in cases of fainting from particular odors, or, in individuals of exquisite musical organization, of syncopal tendency from continued false notes impressing the ear; acting through the uterine nerves, the movements of the fœtus in utero; through the nerves of the stomach, indigestible food,[4] or a draught of cold water taken during perspiration; excessive hunger or nausea; through the cutaneous nerves, injuries to the surface, as burns; through the nerves of the urethra, the introduction of a bougie—may all induce syncope.

Intense emotion of all kinds, pain, the sight of injuries inflicted either on oneself or on others, occasionally arrest the heart's beat.

395. *Hæmic.*—Here appear actual loss of blood, acting both directly on the heart in consequence of deficient immediate stimulus to the ganglia and substance of the organ, and indirectly through impaired innervation; also the circulation of imperfectly oxygenized blood, as where syncope occurs from non-ventilation in crowded rooms; draining of the water, as of other elements, of the blood through profuse discharges, diarrhœal or other. Under this head may also be placed cases of fatal syncope from coagulation within the pulmonary artery—forming a link with the following group.

396. *Organic and Mechanical.*—Here may be ranked cases of fainting from pressure of blood thrown into the pericardium from rupture of the heart, or of an aneurismal sac (the nervous shock of the injury playing a part too in the result); from disturbed circulation depending on ruptured valve, or even on old standing organic disease of a valve,[5] or on sudden obstruction of a coronary vessel;

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[1] From experiments made many years ago, Sir B. Brodie drew the inference that the nicotine of tobacco acted specially on the nervous system, the essential oil on the heart.

[2] The salts of potash, magnesia, zinc, copper, and lime injected into the blood by Dr. Blake appeared to destroy life by specially annulling the heart's irritability.

[3] Harmonizing with the results of Legallois' experiments, which show that, though the heart continues to beat after gradual and piecemeal removal of the entire brain, its movements are arrested by sudden crushing of the encephalon.

[4] In part, however, this may be from the mechanical effects of distension with gas. New bread rapidly eaten, especially if the stomach has been for some time empty, may actually induce fatal syncope. Every one knows the story of Otway's death from swallowing a roll when in a state of starvation.

[5] The only example, strictly in point, of this kind is furnished by the fatal syncope of aortic regurgitation, the mechanism of which will be more fully considered hereafter.

from the entry of air into the circulation; from the sudden removal of extra-pressure, to which it had long been habituated, from a portion of the vascular system, as when fainting follows the incautiously rapid removal of ascitic fluid.

397. As matter of actual experience, syncope is oftener induced by a combination of different causes than by any single one of the number. Fatal failure of cardiac action during the administration of chloroform, in cases where somatic death is very immediately effected through the heart, may be cited as an apposite example. In the first place it cannot be questioned, it appears to me, that in the mass of cases of actual death, or of imminent unachieved death, from chloroform, saturation of the nervous centres, for the tissue of which the hydrocarbons seem to have a special affinity, is the essential and radical cause of the event. Now the medulla oblongata being poisoned, its inhibiting function and that of the vagus being excited, and the heart's own texture being supplied with blood largely impregnated with the inhaled vapor, it follows that anæsthesia of the cardiac ganglia and of the cardiac plexus, with impairment of sarcous irritability, are readily conceivable, nay, necessarily produced, effects. Yet to all these combined conditions death cannot in ordinary cases be referred—for the respiration in healthy animals invariably, or all but invariably, stops before the pulse—the death is asphyxial, not syncopal. It is easier to kill through the medulla oblongata by arresting respiration, than by directly or indirectly annihilating the heart's motor innervation and irritability. Some further influence is wanted, in order that the common sequence of failures shall be inverted, and that cessation of all action in the heart shall precede that in the lungs. Now that further influence is to be found, in the human subject, either in pure and inexplicable idiosyncrasy; or in pre-existing perversion of cardiac innervation; or in actual disease, most commonly fatty metamorphosis of the sarcous or nervous structures of the heart. These seem to be the conditions which occasionally convert paralytic suspension of cardiac movement into the second instead of the third element of somatic death. Nay, more, it is well conceivable that in some instances, where the heart is in a dynamic point of view very exceptionably impressionable [393], or where its proper ganglionic structure has been extensively disorganized, its movements shall be arrested, before even the nervous centres themselves have given evidence of functional disturbance. Death would then be primarily and truly syncopal.

398. *Anatomical Characters.*—Does death by syncope entail, as matter of necessity, any particular state of organs, distinctive of itself, and constituting its anatomical character? I believe this question must be answered in the negative. The mechanism of syncope is so various, its accomplishment sometimes so thoroughly instantaneous, sometimes, relatively speaking, so gradual, that the walls and cavities of the heart cannot be expected to, as they

actually do not, present invariably the same characters. In one case syncope is preluded by feebleness, in another by spasmodic force, of contraction; one individual is gorged with blood, another is anæmic, at the moment of the heart's last beat.

Hence it is that in some cases the muscular structure is thoroughly flaccid, the cavities large and gaping; in others the walls firm and rigidly contracted, the cavities small and closed—the heart's last beat terminating in ventricular systole. So, again, the cavities may be empty; they may contain a small quantity of blood, fluid, grumous or fibrinous; they may contain good quantities of grumous blood. Such blood as is present, is pretty equally distributed to both sides of the organ, or there may be an excess on the right—though never to the degree seen in death by genuine asphyxia. There may or may not be fluid blood or actual fibrinous clot in the pulmonary artery.

Bichat taught that when syncopal death occurs in the course of various diseases indiscriminately, the lungs are "as a constant rule almost entirely free from blood."[1] He ascribes this freedom of the lungs to sudden cessation of the circulation; but in point of fact the cessation is not always absolutely sudden. There is time allowed for stagnating blood to accumulate in the pulmonary vessels. And so, as matter of experience, some slight engorgement may be found occasionally in perfectly healthy lungs after syncopal death; never, however, that massive engorgement of capillaries and fine vessels appertaining to asphyxia.

The encephalon, unless under circumstances wholly exceptional, is pale and bloodless.

399. *Symptoms.*—Syncope, whether induced directly by failure of the heart's irritability, or indirectly through deficient supply or perversion of nervous influence, by loss of blood, or by mechanical causes, occurs with very much the same train of symptoms when actually and completely developed. But in respect of premonitory occurrences and the sequence of symptoms, differences occur according to the nature of the cause.

400. (*a.*) *Premonitory.*—The more important of these are: nausea, sinking feel at the epigastrium, disturbed vision, vertigo, tinnitus aurium, confusion of thought, pallor, drawing of the features, inclination to clammy perspiration, tremulous contraction of the muscles, or slight convulsions, chattering of the teeth, and failure of the pulse. One or more of these may announce the coming syncope; but in a certain proportion of cases the actual stoppage of the heart's action is sudden.

401. (*b.*) *Actual.*—As the syncopal state grows more complete, the pulse, though absolutely wanting at the wrist, may often be faintly felt in the carotids; the patient becomes totally unconscious; the surface cool, clammy, or natural in feel and temperature; the

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[1] "Vie et Mort," p. 189, 3ième édit., 1805.

features contracted; the nares pinched; the lips of marble pallor; the face and skin generally more or less blanched; the respiration suspended absolutely, or almost imperceptible; the pupils dilated: in some few instances the sphincters of the bladder and rectum relax. Meanwhile the heart's impulse may be almost or completely lost to the eye and hand; the sounds nevertheless may still remain distinctly audible; both are, of course, exceedingly feeble, the first very short, the second generally lost at the apex. When the heart's motion wholly ceases, and the syncopal state is carried to its extreme point, no vestige of sound can be caught. Absolute suspension of cerebral[1] and of pulmonary action follows.

Pre-existing murmur may cease to be audible, though the heart's sounds continue perceptible [273].

402. *Duration.*—Lasting for an instant only, for seconds, or for a very few minutes, ordinary syncope terminates by gasping or rather sighing respirations, at long intervals, and gradual return of pulse, consciousness, and color. Sometimes vomiting or discharge of flatus, convulsions, palpitation, or profuse perspiration, take place at the time of returning consciousness. Various paræsthesiæ may be felt in the limbs, and the phenomena of re-establishment of somatic life, are on the whole generally represented by those, who have felt them, as painful and distressing. Yet not always so, if we may trust the quaint and picturesque description left us by Montaigne of his recovery from fainting caused by a fall from horseback.[2]

It would be difficult, if not impossible, to fix with certainty the length of time the heart may fail to give signs of action either to the hand or ear of the observer, without necessarily ceasing to beat for ever. But I do not believe there is any record of trustworthy character proving that its action has ever been re-established after all cardiac sound had ceased for five minutes.[3]

403. *Diagnosis.*—Syncopal unconsciousness will be distinguished from that of *asphyxia* by the pallor of surface; in the latter state congestion and general lividity of the head and face exist, and the heart continues, though feebly, to beat.—In *uræmic coma*, or semicoma, there is pallor of face to deceive, but it is an œdematous pallor; the odor of the breath is urinous; and, finally, the state of

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[1] The ancient synonyms of syncope, *deliquium animi* and *leipothymia*, show the importance of this element of the symptomatology.

[2] . . . Mais à la vérité non seulement exempte de déplaisir, ains meslée à cette douleur que sentent ceulx, qui se laissent glisser au sommeil. . . . Je prenais plaisir à m'alanguir, et à me laisser aller. ("Essais," liv. ii. chap. vi.) In the same chapter the acute essayist expresses the opinion that in all probability the moment of passage from life to death is unattended with travail or suffering of any kind—an opinion much more accordant with observation than that subsequently tendered by Buffon, who romances not a little concerning that "most sharp pain which precedes fainting or death."

[3] Josat peremptorily affirms, that the absence of auscultatory signs of cardiac action, even if "prolonged for some minutes," is not an "infallible sign of death." ("De la Mort et de ses Caractères," p. 81.)

the heart's sounds and the fulness of the pulse at the wrist will prevent error.—*Apoplexy*, though traced *post mortem* to cerebral hemorrhage, sometimes occurs with pallor of face; the state of the pulse and of the heart prevents the possibility of error—in apoplexy, neither heart nor pulse fails in strength, and both may act with undue energy.—*Hysterical trance or lethargy*, with absolute motionlessness, closure of the eyes, almost complete suppression of respiration, simulates syncope; but there is no pallor, and the pulse beats steadily and with distinctness.

The only serious difficulty consists in diagnosing with certainty that rare form of prolonged syncopal trance, in which somatic, and ultimately even molecular, death may be closely simulated. This involves the question of the signs of death.

The alleged characters of real death may be enumerated as follows. Absolute immobility of the body; cooling of the surface; muscular (cadaveric) rigidity; opacity of the tips of the fingers, examined opposite a strong light; lividity and staining of dependent parts; an odor *sui generis;* absence of moistening of the mouth after it has been artificially dried; relaxation of the sphincters; the existence of higher thermometric temperature at a certain depth within the bowel than on the surface;[1] total absence of cardiac sounds; absence of breath-mark on a glass held close to the mouth; non-formation of blebs under the action of boiling water dropped on the skin;[2] non-appearance of blood from incision of a mucous membrane; insensibility of the muscles to the galvanic current; state of flexion of the thumb within the flexed fingers; flattening of the cornea; change of form of the pupil, from the round to the elongated, by lateral pressure on the eyeball (Ripault); dilatation of the pupil, with insensibility to the galvanic current; disappearance (beginning with the deepest) of the three images, which are during life visible in the depths of the eye, if a lighted candle be held before that organ (Legrand).

The conscientious analysis of M. Josat (*loc. cit.*) has shown that no single one of these characters is worthy of implicit trust; but in proportion to the number of them associated in any instance, may the certitude of decease be assumed. Still, in doubtful cases, before a positive opinion is hazarded, sufficient time should be allowed for the development of putrefactive changes; lest a catastrophe, the most horrible the mind can conceive, occur.

The feats of the Hindoo Fakirs, who, it is averred, bury themselves alive, and live in a state of "self-hypnotism" for a considerable number of weeks without oxygen, seem to me to rest on no trustworthy evidence. Even were disinterested witnesses prepared to vouch for the fact, I should avail myself of Hume's argument concerning miracles in general, and prefer believing that a certain number of honest persons had been deceived, than that the laws of nature had been reversed. The raising of a mango tree from the seed in a single hour—stem, leaves, flowers, fruit—a prodigy daily accomplished by Hindoo operators, is a sufficient evidence of the remarkable powers of deception possessed by these people. (*Vide* Appendix.)

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[1] For the account of an instrument invented for ascertaining this character by M. van Hengel, vide "Gaz. Médicale de Paris," 1848.

[2] It appears certain, however, that blebs will sometimes form after real death.

404. *Prognosis.*—In the immense majority of cases syncope is recovered from, and without sequential ill effect. Nevertheless, if chance brought the physician in presence of an attack of syncope in a person of whose medical history he knows nothing, and more especially if that person were no longer young, a cautious opinion as to the issue should be given. Organic disease of fatal tendency may in truth be present, but undiscoverable, on account of the heart's quasi inaction, or but feeble effort at pulsation. It is affirmed that syncope has sometimes terminated fatally in cases of pure anæmia; and inasmuch as I have seen such anæmia end by death, I can well conceive that its immediate mechanism may be syncopal.

On the other hand, syncope, instead of destroying, proves the very means of preserving, life under certain special circumstances. The fatal termination of grave hemorrhages, traumatic and idiopathic, is very frequently prevented by failure of the heart's action giving the necessary time for the stagnating blood, within the bleeding vessels, to coagulate and close their opened orifices.

405. *Treatment.*—In treating an attack of syncope, the first measures are to place the patient horizontally with the head on, or below, the level of the shoulders, to allow a free circulation of cool air, and remove all pressure from the neck and chest. If the syncope be caused by loss of blood, a tourniquet may be applied with advantage to one or both femoral arteries. Stimulant impressions on the nerves—on those of the nostrils and lungs by ammonia, strong acetic acid, the fumes of burning feathers—on those of the skin by the cold water dash, the application of vinegar to the temples, slapping the palms of the hands or surface generally, frictions with stimulant liniments along the spine—on those of the stomach (if the patient can swallow), by a draught of cold water, frequently arouse the heart instantaneously. Ether, aromatic spirits of ammonia, or brandy, should be given internally—if possible, by the mouth; if this be impossible, and the fit be prolonged, enemata, with ammonia, turpentine, or brandy, may be administered. It is scarcely necessary to say, that if protracted syncope depend on an overloaded state of the stomach, an emetic should be given (by the rectum, if otherwise impossible): flatulence may be relieved by the rectum-tube, and an assafœtida and cajeput enema.

406. In cases of protracted fainting, assuming the serious character of suspended animation, hot applications, sinapisms or turpentine fomentations, to the heart and spine, electro-galvanism, and artificial respiration, must be successively had recourse to. If the

cause be loss of blood, transfusion presents itself as a final measure. The last three agents require special consideration.

407. Valentin, Henle, Budge, and others, have distinctly shown that the heart's action may be re-excited after stoppage by galvanic stimulation of the roots of the spinal accessory nerve, and of the first four cervical nerves, and of the sympathetic filaments belonging to the organ. Schiff states that increase of the heart's vigor takes place under very gentle stimulation of the pneumogastric. The recent experiments of Mr. Lister[1] seem to establish the general conclusion, that very mild galvanic currents passed from the posterior region of the spine, in the course of the vagi, and through the cardiac sympathetic excite, while strong currents exercise an "inhibiting" influence upon, the heart's actions. But when exhaustion has occurred in the animal experimented upon, a much stronger current is required to stimulate the heart, than when its nervous energy is scarcely or not at all impaired.

The practical guidance seems clear as to the force of galvanic currents to be employed in cases of protracted syncope in the human subject. The gentlest amount of galvanic stimulus should first be tried: but it is clear, if there has been much previous exhaustion, a very powerful current, which under ordinary circumstances would have arrested the heart's play, may be required to revive it. Caution is clearly of deep importance; Dr. Brown-Séquard is doubtless correct in his strong assertion, "The best means we possess for *destroying* the remains of life in dying persons, is to subject their nerves and their muscles to the *exciting* and *exhausting* action of galvanism." So, too, a Committee of the Société d'Emulation de Paris found that electricity, employed as a general nervous stimulant, rapidly exhausted the nervous excitability of animals in the last stage of chloroformization.[2]

408. Transfusion of blood has been mentioned as a final remedy when suspended animation depends on excessive hemorrhage. The propriety of the measure under these circumstances is self-evident. But, possibly, even in prolonged syncopal trance of other mechanisms, the injection of blood might stimulate the vaso-motor system of nerves, and so act on the heart. The marvellous effects of saline injections in cholera Asiatica, suggest the possibility of their proving yet more useful in syncopal loss of animation. Again, a patient, quasi-dead from cholera, may be temporarily resuscitated by the injection of a fluid imitative in temperature and saline constitution of the water and salts of the blood; true, in the great majority of cases he eventually perishes; but here there is the disease to destroy —in the case of revival from mere trance there is none.

409. In employing artificial respiration gentleness is of the last importance. I long since pointed out the danger of producing intralobular emphysema by too forcible insufflation.[3] Force is fatal.

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[1] Royal Soc., Aug. 13, 1858.

[2] Union Médicale, 1835.

[3] Cyclop. of Surgery, art. Emphysema, p. 79, 1842.

And in truth the efficacy of artificial respiration in genuine syncopal trance must be limited enough. It is another matter, where syncope is secondary to asphyxia, as in cases of imminent death from chloroform inhalation.

B.—Habitual Feeble Action.

410. Persistent impairment in the vigor of the heart's contractions attends a variety of organic changes of its texture, acute and chronic. But such impairment is also observable in some cases, where no appreciable failure or perversion of cardiac nutrition can be detected; and to these only is reference intended in the present place.

411. *Symptoms.*—Where the heart is habitually feeble in action, coldness and clamminess of the extremities, œdema of the ankles and insteps, shortness of breath, frequent inclination to faintness, sensations of languor and ennui; low spirits, anorexia or depraved appetite, foul breath, and constipated bowels, are more or less constant symptoms.

Physical signs.—The heart's impulse is feeble; its sounds want tone. Reduplication of the diastolic sound at the base is common, and palpitation easily excited.

412. *Treatment.*—This state of things, which I have principally seen in young females, and often in connection with disordered menstruation, is curable by attention to the state of the uterus, and by the tonic invigorating plan of treatment. Moderate walking exercise is essential, though commonly very much disliked by the patient. Horse exercise proves serviceable also.

IV.—ANGINA PECTORIS.

413. By the phrase angina pectoris, or "suffocative breast-pang," is understood a paroxysmal neurosis, in which the heart is essentially concerned. As far as it goes, this definition is, I believe, unassailable: its logical defect is inadequacy; a defect that cannot be got rid of, until our notions of the intimate nature of the affection acquire much greater solidity than they at present possess.

414. *Symptoms.*—The dominant symptom of angina is pain; without this, the complaint cannot be said to exist.

The essential seat of the pain, in the great majority of cases, corresponds to the præcordial part of the lower sternal region—as far as mere topography goes, it seems, consequently more directly connected with the right than the left side of the heart. By some exceptional persons, the pain is especially referred to the middle, or even upper, sternal region.

Associated with this pain, appears tenderness in the spot—cutaneous, or at least superficial, hyperæsthesia. There is no positive evidence of increased tactile sensibility of the heart itself.

In character, the pain is rarely dull or aching; much more commonly gnawing, tearing, lancinating, cramp-like, or indescribable—an exquisite torture, grasping, constrictive, and suffocative.

Synchronously with the cardiac seizure, or a little later than this, there may, or may not, occur (the affirmative by far the more commonly) various painful sensations in other parts. Pain shooting to the mid-dorsal spine, to the left side of the neck and occiput, the left shoulder, the entire length of the left arm, or stopping short either at the insertion of the deltoid, or at the bend of the elbow, or limited to the middle inner aspect of that arm, or similarly affecting both arms, or much more rarely the right arm alone, or sometimes passing to the left leg, sometimes invading the four extremities at once, and occasionally involving even the testes—pain thus localized, possesses the shooting, stinging, tingling, and benumbing qualities of neuralgia. These pains are habitually regarded as secondary—reflex, or sympathetic offsets from the cardiac nerves primarily involved.

415. The seizure, in the majority of instances perfectly sudden, occurs without the slightest warning; in rare instances the habitual sufferer receives notice of the coming fit through certain cardiac paræsthesiæ [321]. He knows also that certain movements and acts will either positively, or nearly so, produce an attack.

416. Nothing can be more contradictory than the statements of writers concerning the state of the pulse in angina; cases free from *obvious* organic disease of the substance and of the valves of the heart have evidently not been discriminated from others in which more or less grave lesions of the kind existed. It is, further, very necessary to separate the pulse of the seizure from that of the interval.

Now the pulse in the interval, as far as I have seen, possesses no special character; in frequency, and in force, and in rhythm its attributes are those of health—provided the disease be either ostensibly dynamic, or there be no obvious structural change present, capable of accounting for perversion in one or more of those characters. I have very rarely seen a patient in the actual height of an anginal paroxysm; but that towards its close, in cases of recovery, there most certainly need be neither acceleration nor irregularity of beat, I can aver from my own experience. Angina resembles epilepsy, in that the actual fit is relatively seldom witnessed by the medical observer; still, cases have been recorded showing that in the very extremity of the pain the pulse may be perfectly regular, and scarcely exceed by half a dozen beats per minute the rate normal in the individual—an increase of fifteen to twenty beats has, on the other hand, been observed.

In fits destined to terminate fatally, the pulse becomes almost imperceptible for some moments before death, and may or may not be disturbed in rhythm.

417. The physical signs connected with the heart, provided no obvious organic disease exist, are not necessarily abnormal. The

impulse and the sounds may be natural—there is no murmur. If there be notable disturbance in any of these points, that disturbance is the exponent of pre-existent organic disease. Even rhythm is not of necessity disordered—except where such disease is present. All this is *à fortiori* true in the intervals of seizures: a past attack of angina leaves no impress on the structure or actions of the organ.

418. Dyspnœa, subjective or objective, is not an essential phenomenon of the anginal fit. The number of respirations per minute may *secondarily* be augmented—though never to any notable degree: but a real sense of dyspnœa—a marked increase in the craving for breath—does not occur in the pure disease. Special hyperæsthesia of the medulla oblongata or pulmonary branches of the vagus is not, in other words, excited. It may, on the contrary, almost be affirmed that the special sensibility of the pulmonary nerves is temporarily maintained below par; for the *amount* of expansion with each inspiration is lessened, by combined consensualism and volition, from the dread of increasing the post-sternal pain, while the *number* of breathing acts in the minute scarcely undergoes any, and certainly not a commensurate, increase. By an effort of the will, if the patient have the courage to risk the chance of a moment's extra agony, the chest may be freely expanded. Nay, more, there are exceptional cases wherein, as long since noticed by Parry, momentary release from suffering is obtained by taking a full inspiration, throwing the shoulders back, straightening the spine, and keeping at the "top of the breath."

It may safely be assumed that in cases of angina, where marked lividity of the face and genuine dyspnœa exist, the orifices or walls of the heart are gravely unsound.

419. The gastric branches of the vagus are not always implicated. But violent and continued eructation (not explicable by the nature or recency of the last meals) and sometimes vomiting, every now and then, prove the existence of irritation in those filaments. Flatulent distension of the entire abdomen is sometimes observed.

420. The encephalic functions remain unaffected—one of the most distressing parts of the seizure arises from the clearness of the perceptive faculties.

421. Slight convulsions occur occasionally; the gravest tetanic spasms, with complete opisthotonos, have been witnessed. When the seizure has been of this kind, tonic spasm may hold on, in some spots, hours after the actual fit has passed away.[1]

422. There may be a full discharge of watery urine.

423. *General Symptoms.*—The general symptoms are those of nervous shock—pallor of surface, cold clammy perspiration, fall in temperature of the surface generally, intense anxiety of countenance, and not only dread of approaching dissolution, but profound conviction that life cannot go on unless instantaneous relief be afforded.

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[1] Allingham, U. C. H., Females, vol. xvii. p. 57.

424. *Terminations and Duration of the Fit.*—Lastly, a few seconds, or a few minutes, the anginal seizure may continue to recur, with variable intensity, several times during an hour or more; a paroxysm may thus be single or composed of a number of successive seizures. In either case, if the issue be favorable, it goes as suddenly as it came—leaving as its sequence a feeling of general exhaustion, accompanied with a strong sense of present relief. Or death takes place in the manner to be presently described.

The termination of the attack is sometimes followed by temporary hydruria.

425. *Manner of Death.*—The immediate mechanism of death appears, as far as evidence goes, to be syncopal. But the conditions, directly antecedent, vary. Thus, the fatal syncope may be perfectly sudden, instantaneous, coëval with a single pang—there may be no time for struggle, for pulmonary congestion, for external convulsion: the most perfect placidity of expression, and calmness of attitude are under those circumstances found in the corpse.[1] Or death may be a more deliberate process: the pulse gradually fails; fits of brief duration follow each other rapidly; anxiety and dread of dissolution reach their possible limit; cold clammy perspiration breaks out on the surface; the radial pulse becomes imperceptible; the breathing grows labored; trismus with clonic spasms of the limbs, or it may be of the entire trunk, occurs; insensibility follows, and in this state the sufferer rapidly perishes.

426. *Recurrence.*—Angina belongs essentially to the class of recurrent affections—the fact of one seizure having taken place is a reason why others should follow. And with each returning seizure the activity of the directly exciting cause is generally noticed to have lessened—so susceptible does the nervous plexus concerned become, that an influence which at first would have failed to excite, eventually readily produces a paroxysm. The first seizure generally comes of effort of some kind—more particularly that of walking briskly up a hill or even on level ground. Sauvages (1763), exaggerating the importance of this character, makes its occurrence a *sine quâ non*, by giving it a prominent place in his definition of the disease.[2] The patient, forced by the agony to stop, is at once released—steps forward again, is again brought to a stand still: and this process having been repeated a variable number of times, walking becomes possible—it may be even with enjoyment; or, either from dread of the pain, or from real return of it every time the effort is renewed, the attempt is relinquished.[3]

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[1] In a case of this species, where death occurred instantaneously while the patient was reading quietly in bed, there was so little disturbance of the frame that his thumb and forefinger were found in the pamphlet on which he had been engaged; the bedclothes, too, were undisturbed. (Case seen with Mr. G. Bird.)

[2] Dr. Latham records a case in which the first seizure took place in bed.

[3] "I can walk with ease ten or fifteen miles" (said a patient to me, *within three days of his death in a paroxysm*), "after I have been stopped three or four times at

Now, a paroxysm of this kind may, it is affirmed, remain single for a lifetime of ordinary duration, provided all conceivable exciting causes be carefully avoided. I have known instantaneous death occur from angina in a male, aged about forty-five, who had but three fits—the first one year, the second half an hour, before the final and fatal one.

But such distant recurrence is not the rule—the intervals of freedom gradually or rapidly shorten; and eventually emotion of any kind, sudden movement, the acts of defecation, of coughing, of quickly swallowing water, will induce a fit; or no appreciable cause of any kind may precede. Attacks occasionally, but certainly not commonly, take place as the patient drops off to sleep. It is said the returns are sometimes distinctly periodical—and that this is one of the numerous forms malarial neurosis may assume.

427. *Duration of the Disease.*—In regard of this point the greatest diversity prevails. On the one hand life may be prolonged for years after the first seizure, and in spite of more or less frequent recurrence. At the other extreme, as matter of recorded fact, must be placed a case published by Dr. Latham, where death took place in about two hours after the first outbreak of pain. Now this case justifies us in supposing that the total duration may be yet briefer—and that, occasionally, instantaneous and unwitnessed death, occurring in persons previously believed to be healthy, has been accompanied with the phenomena of angina. Be this as it will, there is no doubt that in the generality of instances the complaint runs a somewhat protracted course. Data are wanting wherefrom to calculate the mean duration.

428. *Anatomical Conditions.*—First, there are few, if any, structural diseases either of the heart, its orifices, and its nutrient arteries, or of the aorta, which are not to be found recorded in the narratives of the *post-mortem* examinations of different victims of angina pectoris. Secondly, there is no conceivable disease of those structures and parts which has not in various individuals reached the highest point of development, without anginal paroxysms, even of a slight kind, having occurred during life: to this proposition extensive calcification of the coronary arteries, perhaps, furnishes a solitary exception. Thirdly, the organic changes most frequently met with have been fatty atrophy and flabby dilatation of the heart; obstructive disease of the coronary arteries by atheroma and calcification; and calcification of the orifice and arch of the aorta. Fourthly, the rarest changes have been hypertrophy and hypertrophy with dilatation. In truth, it may be doubted whether these conditions in their genuine form, without any combination of fatty atrophy, have ever been the sole morbid states present.

Now the conclusions from these premises cannot be other than that, even in the most serious case of textural change, there is

intervals of a hundred yards." This person succeeded well in walking up hill *zig-zag and sideways.*

neurotic disturbance beyond that change as the essential element of the paroxysm;—that textural change acts simply as a promoter of the seizure, by rendering the nerves in some way more apt to suffer dynamic disturbance;—and that the forms of organic disease most favorable to that disturbance, all of them, indicate mal-nutrition and weakened power in the parts concerned.

If we must place implicit faith in all recorded observations, we are called on to admit that death may be produced by angina, the heart and vessels being texturally sound. I confess I cannot help doubting this. Recent narratives, as far as I know, invariably describe some organic change; and older accounts are not trustworthy, seeing that accurate knowledge concerning dilatation, and various forms of mal-nutrition has been the acquisition of the last few years only. My own experience, as far as it goes, emphatically deposes to the constancy of structural disease. It has occurred to me to examine during life some twelve or fourteen cases of thoroughly well-characterized angina; in every one there were physical signs of organic change—flabby or fatty dilatation and aortic disease principally. I have opened, or seen opened, the bodies of four persons destroyed in the actual paroxysm; the heart or aorta, or both, were texturally affected in all.

429. The appearances dependent on the manner of death are among those of syncope. Here were, for instance, the essential conditions I found in the body of a male, aged sixty, cut off in the paroxysm:—

Twenty-six hours after death; weather cold, damp; *cadaveric rigidity extreme;* vibices posteriorly abundant; subcutaneous thick layer of fat; face pale, without lividity; no fluid in either pleural or pericardial sacs; old adhesions posteriorly, dark congestion moderate anteriorly, both lungs; large mass of fat attached to edge of base of right lung; much sub-pericardial fat; *heart very flabby, may be rolled up,* not notably enlarged as a whole; walls neither specially thick nor thin, except about right apex, where the sub-pericardial fat has encroached on the muscular substance; *a little fluid blood in four cavities, no coagula; some fluid blood in pulmonary arteries, no embolus;* no valvular disease of any kind; *very moderate atheroma and calcification of coronary arteries;* aorta, opened in entire length, calcified extensively, with minute superficial fissures here and there. *Very partial and incipient fatty atrophy of heart's fibre.*

The total absence of firmness, and of any indication of spasmodic contraction, of the heart (while cadaveric rigidity existed in the external muscles to a very marked amount) is worthy of note—also the almost complete absence of blood from the heart's cavities, and the freedom of the pulmonary vessels.

430. *Predisposing Causes.*—Angina predominates vastly in males; the ratio of ten to one is deducible from some published estimates.

The disease is rare before the fiftieth, excessively so before the fortieth, year,[1] and unknown in infancy and childhood.

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[1] Of 88 cases, collected by Sir John Forbes, 80 refer to males; and of 84 patients, 72 had passed the fiftieth year.

Angina is certainly very materially more common in the better than in the humbler ranks of society; and my own experience, confirming that of Sir Gilbert Blane,[1] scarcely supplies me with more than a solitary well-defined example of true angina in hospital practice.

I have seen a case or two leading me to surmise that spanæmia, where other conditions are favorable, will promote the occurrence of seizures.

Laennec supposed that epidemic influence renders the disease unusually common from time to time.

431. *Diagnosis.*—Whether an intra-thoracic pain is really anginal proves, as a general rule, not difficult of determination. The intensity and locality of the suffering, and dread of impending death, the circumstances of movement under which the original seizure commonly arises, suffice to distinguish it. Still the affection is occasionally simulated more or less closely by some one of the conditions I have grouped under the heading of Pseudo-Angina, to the description of which I refer for the means of distinction.

Admitting a given case to be one of angina, it remains, as a matter of serious importance, to ascertain the structural conditions of the heart. The presence or absence of far the greater number of organic diseases can easily be determined by the rules given with the account of each of the series. But it unfortunately happens that the two organic affections most commonly associated with angina, namely, fatty atrophy of the muscular substance, and calcification of the coronary arteries, are precisely those least susceptible of sure diagnosis. Demonstration of the latter condition lies beyond the limits of existing knowledge wholly: and of the great difficulty attending the detection of the former, unless in its very aggravated forms, enough is said with the account of the disease.

432. *Prognosis.*—The life of him who has had an unmistakable attack of angina is not insurably safe for an hour. And yet existence *may* be protracted for years. Is there any method whereby we can with a reasonable amount of security place a given sufferer among those who shall be cut off perhaps the next instant, or among those who shall survive for years? I know of none. The presence or absence of demonstrable organic disease and the quality of this, so far from guiding us correctly, may prove the source of complete error. I have examined a man in whom nothing physical could be detected except feebleness, by no means great, in the cardiac action, who perished forty-eight hours later in the paroxysm—post-mortem examination proving the absence of any structural change except moderate softness of fibre with a very slight, almost problematical, amount of intra-sarcolemmous fat. And *per contra* it has occurred to me to treat a certain number of cases, where, in

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[1] Med.-Chir. Trans., vol. iv.

spite of different forms of obvious organic mischief, life has held on for months, though paroxysms have all the while more or less frequently recurred. Hence in my mind there is great fallacy in the doctrine that so-called "functional" angina ("functional," perchance, solely because textural change eludes detection) is a relatively safe complaint, and "organic" angina alone pregnant with constant danger. The cardinal fact in the prognosis of real angina under any circumstances is its uncertainty.

433. *Nature, seat, and mechanism.*—That angina pectoris is in its essence neurotic, appears from the sudden advent and departure of the seizures—from the character of the attacks while actually present—from the perfect ease enjoyed in their intervals—from the impossibility of assigning to the affection any special anatomical character—and from the kind of treatment that proves beneficial.

Now pain is obviously enough the essential neurotic element of the disease. As to the nerves involved, the statements made concerning cardiac neuralgia seem distinctly applicable [313]. Plainly the pain has nothing to do with the chest wall—it is neither cutaneous nor intercostal: it is at the least intra-thoracic; and the final influences it exercises on the functions of the heart in fatal cases, as well as the manner of its radiations,[1] localize it in the cardiac plexus. The special proneness of these nerves to suffer arises possibly in all, certainly in the great majority of cases, through the disturbing influence of structural changes in the heart.

The mechanism of the neurosis seems centric, reflex, or intrinsic, according to circumstances. We can scarcely doubt centric origin, when the paroxysm is excited by anger or other strong emotion:[2] when it follows the ingestion of a full meal, or the blowing of wind on the face, we can scarcely question the reflex agency of the gastric vagus and the trifacial: when the agony instantly succeeds effort or abrupt movement, the direct influence of the circulation through the heart's texture on its own nerves seems clearly indicated.

But with this pain, its essential element, does any other neurotic disturbance co-arise in the paroxysm? Heberden and Dr. Latham espouse the doctrine of cardiac spasm—but whether this spasm be the result of the pain, or, as ably argued by the latter observer, its cause, has been admitted to be an unsettled point. Parry and Dr. Stokes, on the ground of clinical observation, look upon temporary increase of weakness in an already weakened organ, as the essential muscular element of the anginal paroxysm.

For my own part, I regard the presence of spasm, either initiative or sequential, either simple or with cramp, as altogether doubtful in the ordinary class of cases. In the first place it appears to me incontestable that anginal suffering may exist, and in a well-developed form, without spasm. For how is it possible that a heart spasmodically contracted can act rhythmically, as all good observers admit the organ habitually does, where no *organic* disease, of necessity influencing the healthful measure of the pulse, coexists? And if the seizure may thus be pushed to the imminence of death without spasm, it seems unlikely the mode of action should change just at the moment of the fatal syncope.

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[1] Vide Mueller's Physiology, by Baly, vol. i. p. 666.

[2] I accept as granted that the immortal Bichat erred in the doctrine he so firmly espoused, as to the direct influence of the emotions on the nerves of the heart, to the exclusion of any portion of the great nervous centres.

I cannot help believing it most probable that the pain, when it gravely affects the motor innervation, does so paralytically—that the death is one of suspended and not perversely excited contraction. When the pulse does suffer change, its dominant condition is increasing weakness: besides, in the few cases I have opened, the heart bore no marks of recent spasm—it was flaccid and yielding. And, further, who ever meets with genuine angina in the possessor of a strong purely hypertrophous heart?

But, while holding on these grounds very firmly to the doctrine of paralysis, as the perverted motor state in ordinary angina, I am not prepared to affirm there may not be exceptional cases in which simple spasm, or spasm with dislocation or rupture of fibre (true cramp) really occurs. I would suggest at least that in those rare instances where intense tetanic spasm invades the external muscles, an analogous state of the heart may be the real cause of its stoppage; but I should not, even so, be disposed with Dr. Latham to look on the pain as the effect of the spasm, but to view the spasm as the reflex disordered motor result of the pain.

It is not unworthy of note how excellently the phenomena of angina occasionally contribute to demonstrate the independence of the nervous forces severally exciting mere muscular contraction and rhythmical motion of the heart: while the former is gravely and increasingly affected, the latter may maintain the even measure of health almost to the last moment of existence. Pathology might, here, as in so many other instances, have served as a guide to physiology.

434. *Treatment.* (*a.*) *During the fit.*—Sedatives and stimulant antispasmodics are the medicines essentially to be trusted to during the fit. The dose of opium will be measured by the intensity of the pain; from forty to sixty drops of laudanum or of the liquor opii sedativus may, in a severe case, be given along with brandy or from half a drachm to a drachm of ether or aromatic spirits of ammonia, and repeated according to the urgency of the suffering. Musk, camphor, and belladonna are of very inferior importance. Mustard poultices may at the same time be applied to the heart and to the dorsal spine, or cloths wetted with the strong liquor of ammonia laid upon the præcordial surface. Laennec's suggestion of the transmission of a magnetic current (with or without acupuncturation) through the chest, has scarcely been fairly tested. An electro-galvanic current, however, affords better chance of successful influence, and in a serious case of quickly recurring paroxysms,

which seemed indisposed to yield to the ordinary sedative management, deserves trial. Of the necessity of cautious proceeding in this trial enough has elsewhere been said [407]. The mustard pediluvium, especially if the patient be gouty, is useful; the application of chloroform to the præcordial region hazardous. Inhalation of small quantities of ether has been recommended; I have no experience of the practice.

Speciality in the circumstances of the attack may call for special treatment. If the patient be the subject of undoubted sthenic plethora, and especially if the heart be known by previous examinations to be a well-nourished one, the abstraction of blood from a vein, or by cupping between the shoulder-blades, is clearly indicated; but bleeding must not be heedlessly undertaken and without assurance as positive as is attainable, that the heart is at least not a dilated, soft, or flabby one. If flatulence and acidity exist, soda, cajeput oil, and sesquicarbonate of ammonia may be administered with the opiate medicines; if a large undigested meal lie in the stomach, it should at once be removed by an emetic of sulphate of zinc.

435. (*b.*) *Prevention of fits.*—A paroxysm, of which the too experienced patient learns by his feelings to expect the approach, may sometimes be averted completely by an opiate; and sufferers should always carry, properly protected, on their persons, an antispasmodic and sedative draught. One of the best safeguards against seizure consists in the idea, that the means of averting evil consequences, if it occur, are within reach.

436. (*c.*) *In the intervals.*—A person who has had one attack of angina pectoris must remember, that instances, in which recurrence does not take place, are altogether exceptional, and further, that the periods of recurrence gradually approximate more and more, while each successive paroxysm, as a rule, exceeds its predecessor in severity. If it be true, as we have seen, that a first attack is generally brought on by an effort of some kind, such as walking up a hill, or in the teeth of a sharp wind, on the other hand, the most trivial influence will eventually suffice to produce a paroxysm; emotion of any kind, sudden movements of the trunk or arms, efforts at defecation, the acts of coughing, drinking rapidly, &c. Hence, it is clear, the subject of angina must live according to the most stringent rules; every conceivable precaution must be taken to keep the heart in a tranquil state. The patient should give up exciting pursuits of all kinds—intellectual, corporeal, and emotional—and learn, if he be of the *genus irritabile*, to govern his temper. Daily exercise should be slowly taken on perfectly level ground, either on foot or in an easy carriage; riding on horseback is scarcely to be permitted with safety. The diet should be moderate in quantity, simple in quality; the bowels never allowed to be confined. A belladonna plaster worn over the heart, and an issue, seton, or perpetual blister to the arm, have appeared useful in some cases; if

the patient have confidence in counter-irritation, this should by no means be neglected. Change of scene and travel, coupled with the use of tonics, vegetable or mineral, will, by improving the general health, render the patient less prone to seizures. Arsenic, nitrate of silver, and sulphate of zinc are the best of the class of mineral tonics, unless anæmia be present, when, of course, iron is *the* remedy. The removal of gout, chronic rheumatism, or old-standing skin-diseases should be very cautiously, if at all attempted, in the subject of angina: relief of those complaints is unquestionably sometimes followed by increased severity of the cardiac affection.

If angina of malarious origin and periodical recurrence really exist, its treatment would be that of miasmatic diseases generally.

V.—PSEUDO-ANGINA PECTORIS.

437. Genuine angina pectoris is undoubtedly a very rare affection. On the other hand, I very frequently meet with a form of complaint combining in a minor degree many of the characters of angina; and to this imitation of the true disease I propose to give the name of pseudo-angina. I believe that herein lies the explanation of Laennec's notion (so discordant with the experience of English observers), that angina pectoris is of very frequent occurrence.

438. The *symptoms* of pseudo-angina are, more or less severe, commonly constrictive, pain referred to the region of the heart, palpitation occurring paroxysmally without obvious cause, or under exertion, or through over-eating, indigestion, flatulent distension of the stomach, and a variety of other functional disturbances. The breathing becomes panting and suspirous. Giddiness and faintness are sometimes observed. The patient dreads efforts of all kinds.

439. Physically the heart may be to all seeming healthy. The apex beats in the usual spot, and with natural characters; even in the height of the paroxysm no murmur of any kind exists. Shortness, feebleness, and heightened pitch of the first sound at the apex are the only conditions at all frequently met with. These indicate tendency to dilatation, and weakened power: the impulse sometimes wavers and is of uneven force from time to time. But even in cases where all seems physically normal, there may be organic disease beyond the diagnostic means of the present day: if so, however, it must certainly be slight.

440. Like true angina, its imitation is more common, though by no means to the same extent, in males than females, and in the higher than in the lower walks of society; but, unlike true angina, frequently occurs in young adults.

441. Pseudo-angina has fallen under my notice as an attendant on various hæmic diseases—chronic gout and rheumatism, and spanæmia: on nervous diseases—hysteria, spinal irritation, and epilepsy; on intercostal and mammary neuralgia, the direct evidences

of which are more or less complete. Pseudo-angina also occurs in connection with a variety of nervous disturbances, the influence of which can only be made intelligible by reference to some physiological facts. That *suddenly* perverted dynamism of the brain and cord affects the heart's action is proved by the phenomena of simple concussion of those centres; the influence of irritation of the roots of the spinal accessory, of the first four cervical nerves, of the par vagum, of the phrenic and some intercostals, of the cervical ganglia, especially the first, of the cardiac nerve, of the semilunar ganglia and solar plexuses, of the nerves of the stomach, of the hepatic, ovarian, uterine, and even cutaneous nerves, has, in each instance, been proved by more or less precise experimentation. The physiological sympathies of the heart being thus extended, it is not to be wondered that functional disturbances of almost every organ may entail derangement of cardiac action and feeling. Of feeling, for there seems fair reason to suppose that the heart, though unendowed with animal sensibility in health, may acquire it in disease and under the influence of special impressions.

442. *Diagnosis.*—Hysterical and anæmic palpitation, accompanied with intercostal neuralgia, nay, even organic palpitation in a person with that form of neuralgia,[1] may readily be mistaken for true angina; indeed, an attempt has been made by some French writers to show that the latter disease is nothing more than a "brachio-thoracic neuralgia," the heart-symptoms being purely accidental. In true angina the points of tenderness in the course of an intercostal nerve are wanting; besides, the severity of the suffering in the cardiac region is infinitely greater than in intercostal neuralgia.

I have at this moment, under occasional observation, a singular case of a youth who has periodical returns of severe neuralgic pain *originating* at the insertion of both, but especially the left, deltoid muscles: præcordial pain, with slight or grave syncopal feelings, *follows.* There is dilated hypertrophy, with systolic basic murmur, and certain anomalous physical phenomena. But the functional state is not true angina.

Although common sense and experience point to the necessity of learning the cause of pseudo-angina, in order to cure the effect, still, in instances where we either fail to discover that cause, or find it to be itself non-removable, much good may be done to the cardiac symptoms by direct treatment of these on the principles applicable in cases of true angina.

It is in cases of this description that the inhalation of ether or of chloroform may be cautiously allowed.

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[1] Roberts, U. C. H., Females, vol. v. p. 217; Hawkesford, id., vol. v. p. 1.

SECTION II.—ORGANIC DISEASES OF THE HEART.

§ I.—ANÆMIA OF THE HEART.

443. Although anæmia of the texture of the heart has not attracted notice on the part of systematic writers, there can be no doubt of its real existence as a definite morbid state. However, as actually seen, it commonly forms an element of some complicated form of change, to which attention is, excusably enough, perhaps, mainly turned.

444. Anæmia gives a mottled pallor to the cardiac substance; and, if the conditions inducing it have been of long standing, lessens the amount of muscular tonicity and firmness. The softness and pallor accompanying obstruction of the coronary arteries, and the early period of fatty atrophy of the fibre, are examples in point. In extreme general spanæmia the pallor of the heart is striking. Now it is well known that such spanæmia occasionally proves fatal; and the difficulty of accounting for the event has been recognized by Chomel and others. In a case of the kind, where very careful examination, both before and after death, failed to discover any commonly acknowledged cause of death, I was struck with the intense pallor of the heart—and it appears not unreasonable to admit that the slow extinction of the patient, by a sort of protracted syncope, may have been chiefly effected through the gradually lessening contractility of the heart's fibre, fed as this was by imperfect blood.[1] In the case referred to, there was no leucohæmia or enlargement of the spleen; nor were the supra-renal capsules diseased or the skin discolored.

445. In cases where anæmia has either been persistently, or with occasional intermissions, sustained for a more or less lengthened period, it is probable changes are effected in the structure and properties of tissue of the heart. The weakened power of resistance of the ill-nourished muscular substance must, it can scarcely be questioned, facilitate the occurrence of dilatation of the heart's cavities, where efficient extrinsic causes of that dilatation are at work. On the other hand, hemorrhagic spanæmia, even of the gravest kind, will not reduce the resistant force of the walls of the ventricles so much as, unassisted, to lead to such dilatation; the proof of this is, that the hearts of persons cut off by cancer of various organs, though highly anæmic, are not necessarily dilated.

§ II.—CONGESTION OF THE HEART.

446. The substance of the heart, and of its membranes, as of all organs, is subject to three species of congestion, active, passive, and mechanical. There can be no doubt these forms of congestion con-

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[1] Monk, U. C. H., Males, vol. xli. p. 71.

tribute their share, sometimes an important share, to the sum total of functional disturbance occurring in different cardiac diseases. Yet they have attracted little attention from clinical observers, because they are, at least commonly, lost in the graver anatomical changes with which they are associated, and because they lie without the pale of sure diagnosis either through subjective symptoms or objective signs.

I.—ACTIVE CONGESTION.

447. Active congestion forms of course an element of the different inflammations that affect the heart and its membranes. But there seems fair reason to believe that active congestion, at least of the pericardium, may occur independently of the sequential exudation-process. In certain cases of rheumatic fever and of Bright's disease, I have known excited cardiac action, uneasy sensations in the præcordial regions with tenderness under pressure, accompanied with slight, but unmistakable pericardial rub, pass off without any evidence of fluid effusion into the sac. Is it not reasonable to admit (as it is certain vascularity alone may cause friction-sound) that in such cases simple active congestion has occurred and passed away without issue?

And, again, cases now and then occur tending to show the reality of active hydropericardium; active congestion must, if such cases be real, lead the way, preceding the escape of serosity.

II.—PASSIVE CONGESTION.

448. Although it would appear probable the heart's circulation must tend to stagnate during the course of adynamic diseases, the evidence of such condition is wanting. In typhoid (Peyerian) fever, for instance, instead of being abnormally loaded with blood, the minute vessels of the heart seem to contain less fluid than natural. M. Louis insists on the dry look and deficiency of usual moisture on the surface of sections of its tissue.[1]

III.—MECHANICAL CONGESTION.

449. Mechanical congestion of the heart's texture and membranes is of very frequent occurrence.

450. Slightly increased width of the walls of the ventricles, fulness of the great cardiac vein and of its branches and their radicles, undue moisture and deep vinous coloration of the muscular tissue, with minute dark-colored ecchymoses under the pericardium and endocardium, constitute the anatomical characters. The right cavities of the heart are commonly loaded with blood.

451. Obstructed circulation through the lungs, direct pressure either on the large venous trunks supplying the right auricle or on the great cardiac vein, and obstruction to the onward movement of

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[1] Affection Typhoïde, t. i. p. 330, ed. 1.

the blood through the cavities of the heart, are the essential causes of this form of congestion.

Hence it is an attendant on asphyxia, and on capillary bronchitis, especially when superadded to emphysema; is seen in all cases of mediastinal tumor and aortic aneurism, when pressure chances to bear on the vessels just named; accompanies cyanosis dependent on constriction of the pulmonary artery; adds to the mischief in tricuspid regurgitation and constriction; and may even eventually be induced in sequence to capillary stasis in the lungs, by disease of the mitral orifice.

452. I know of no means by which this state may be directly diagnosed; but its existence becomes matter of fair inference under the circumstances just mentioned.

453. The effects entailed by mechanical congestion of the heart will depend on the abruptness or slowness with which it is produced, and on the brevity or length of its duration.

When suddenly effected it seems possible it may induce hydropericardium—as I shall endeavor to show with the history of that affection.

When slowly effected and long maintained such congestion must interfere with the heart's nourishment. This interference, in turn, theoretically lessens the power of resisting the eccentric pressure of the blood on the part of the walls of the organ; hence dilatation of the cavities follows. And as matter of observation dilatation is found with tolerable frequency in cases of pressure of veins by intra-thoracic tumors and aneurisms. True, in aortic aneurism, such dilatation may be wholly wanting, even where the sac springs from the immediate vicinity of the sigmoid valves; but may not this depend, in some cases at least, on accidental deficiency of the pressure in question?

And further, the probabilities are that long-continued stagnation will end in local exudation-process of low type—the effused fibrin, converted into induration-matter, must gravely impair the contractility of the infiltrated parts, and so in another form lay the groundwork of dilatation.[1]

§ III.—ACCUMULATION OF BLOOD IN THE HEART'S CAVITIES.

454. Accumulation of blood may occur in any, or in all, of the cavities, in cases of endocarditis, as a consequence of fibrinous particles interfering with the free play of the valves, and in cases of rupture of valves. Accumulation in the right cavities, especially, will ensue in prolonged fits of palpitation, with gravely disturbed rhythm; in cases of tricuspid regurgitation; during fits of dyspnœa, in highly marked emphysema of the lung, especially if this has already led to organic dilatation of the right cavities; in

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[1] W. Jenner, Med.-Chir. Trans., vol. lii.

capillary asphyxiating bronchitis; in acute pneumonia; and, in a word, to a greater or less extent, in all cases of notably obstructed circulation through the lungs.

455. The symptoms associated with such loading of the right heart are dyspnœa even to orthopnœa, dry cough, venous congestion of the face and upper surfaces generally, unattended, unless there be prior anasarca of the lower extremities, with œdema; oppression, anxiety, and sometimes pseudo-anginal feelings.

456. The heart's impulse, labored and struggling, irregular in force and rhythm, is seen and felt more extensively than natural, especially to the right of the sternum, and at the epigastrium. The area of dulness exceeds that of health, especially about the right costal cartilages.

457. Venesection, cupping over the præcordial region, sharp, rapid purgation, counter-irritants to the lower limbs, or the application of Junod's apparatus, are the remedies theoretically indicated for the relief of this state; practically, too, they prove efficient. But the condition, on which the obstruction of the cavities depends, remains of course in the back-ground, unmodified, or scarcely modified, by them. Ulterior measures must be taken for the removal of that condition, if possible.

In the foregoing brief description, I have had in view the effects of accumulation of fluid blood in the cavities; the phenomena observed, when the clotting-process takes place, are considered under the head of BLOOD-CONCRETIONS IN THE HEART.

§ IV.—CARDIAC INFLAMMATIONS.

I.—PERICARDITIS.

458. Pericarditis, or inflammation of the proper tissue of the pericardium, is clinically known in the acute and chronic forms.

A.—ACUTE PERICARDITIS.

459. *Anatomical characters.*—The anatomical stages of completely evolved acute pericarditis are five: those of dryness with vascularity; of plastic exudation; of liquid effusion; of absorption; and of adhesion.

460. (*a.*) The dry stage is marked by florid, arborescent, and capilliform injection, sometimes by prominent tufts of capillary vessels,[1] especially on the cardiac portion of the sac; the membrane itself is dry and parchment-like. This stage is very rarely seen except where persons, dying of other diseases, are seized with pericarditis, just before dissolution.

461. (*b.*) In the exudation-state plastic lymph of various degrees of firmness accumulates in one or other of the following situations:

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[1] F. Parker, U. C. H., Males, vol. iv. p. 177, Dec. 1848.

on the cardiac and parietal serous surfaces both; on the cardiac division of the membrane only—never, as far as I happen to have seen, on the parietal portion solely; on the posterior aspect only of both cardiac and parietal portions; on the anterior surface of the right ventricle only; or specially about the roots of the great vessels. The lymph is laid down stratiformly, in thick or thin layers; or in lace-like fashion, resembling the intra-cranial osteophyte of pregnant women;[1] or in hillocks; or, like the sand on the sea-beach, in regular layers separated by indentations; or locularly; or in loose thread-like manner. Three-quarters of an inch is the extreme thickness I have witnessed in a layer of lymph—and this only about the great vessels. Essentially of straw color in itself, it is reddened by bloodvessels, developed *de novo* within itself, and by imbibition of blood.

In a fresh attack of pericarditis, lymph will not be thrown out on the immediate surface of old "white patches," whether these be of inflammatory origin or not.[2] And pressure, acting on any particular portion of the pericardial surfaces, will not only prevent the exudation-process, but even active congestion, from occurring at the spot. The best example I have seen of this fact occurred in a case where hydro-pneumothorax on the left side played the part of the compressing agent.[3] This peculiarity is evidently suggestive in regard of the local therapeutics of inflammation.

462. (*c*.) The liquid effusion of the third stage varies in composition and qualities. It may be sero-albuminous and flocculent; sero-fibrinous and spontaneously coagulable after removal from the sac; hemorrhagic; sero-purulent—pus in sufficient quality to be recognized with the naked eye being very rare; or, lastly, it may be wholly composed of pure pus. The latter condition of the fluid is, however, so rare, as almost to take rank among the *mirabilia* of morbid anatomy.

The quantity of liquid commonly ranges between three or four to ten or twelve fluid ounces. But when the amount of plastic exudation is abundant, and when death has occurred very rapidly, only an ounce or two may be found between the meshes of that exudation. On the other hand, where the affection runs a lingering course, enormous accumulation may occur—reaching, in one case which fell under my notice, sixty measured ounces.[4]

The closed sacculi, probably glandular, described originally by Lower, and again recently by M. Corvisart, as seated at the base of the pericardial sac, have not been shown to exercise any definite influence on the effusion-process.

The liquid macerates, softens, rarefies, and so promotes the distension of the sero-fibrous texture of the sac.

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[1] Vide Products, Adventitious, Cyc. of Anat. and Phys., p. 135.

[2] Truman, U. C. H., Females, April 5, 1859.

[3] Plympton, U. C. H., Males, vol. iv., or Clin. Lectures, Lancet, 1849, vol. i. p. 579.

[4] Bartlett, U. C. H., Males, vol. iv. p. 292.

I have never seen actual ulceration or sphacelus of the serous tissue.

Pneumo-pericardium, resulting from chemical changes in the fluid and non-dependent on sloughing of tissue, appears to have fallen under the notice of observers in singularly rare instances.

463. (*d.*) In the absorption-stage serous fluid is removed with ease and rapidity—hemorrhagic effusion with less facility, and pus with still greater difficulty. Exudation-matter of variable plasticity remains behind—to form the material of adhesions, loose or agglutinative, and to furnish a nidus either for the reception of various saline or animalized precipitates, or for the development of sundry pseudo-tissues. In some cases even exudation-matter itself appears to be freely removed by absorption.

464. (*e.*) In the final, or adhesive, stage the two pericardial surfaces may become universally agglutinated together; or locally adherent, either closely by patches or loosely by bands; lateral union of the great vessels is common. Or solidified exudation-matter may exist without adhesion occurring: this is particularly observed about the great vessels, where such matter may long (probably in perpetuity) remain the source of certain important physical signs above the heart's base. Locular or mesh-like adhesions, containing fluid, absorbable and renewable, sometimes form.

465. Thus terminates the disease, anatomically, in its acute stages; any ulterior changes, arising in the tissue or interior of the sac, clinically belong to, and will be described with, the chronic disease.

466. *Secondary lesions.*—Pericarditis, so frequently holding the position of a secondary lesion to other diseases, rarely entails anatomical change in disconnected tissues or organs. The various grave cerebro-spinal symptoms sometimes witnessed are not assignable to any textural alteration in the nervous centres; they are purely dynamic.

Occasionally myocarditis, exudative and suppurative, occurs in the strata adjoining the pericardium; but here we have mere extension of a disease by contiguity of texture. The fibre of the heart sometimes rapidly falls into a state of local atrophy, giving place to intra-sarcolemmous fat—but this can scarcely be regarded as a true secondary lesion. Endocarditis, frequently coexistent, is rather a joint effect of the cause producing the pericarditis, than a sequential result.

I doubt the bronchial inflammation, which occurs in a certain share of cases, being other than an additional effect of one and the same cause. But pneumonia does sometimes arise to all appearance, and pleurisy to a positive certainty, under the direct influence of pericarditis.

467. *Causes and circumstances of origin.*—Pericarditis is rarely, if ever, an isolated disease, and rarely, except when traumatic, springs

up in a person of sound constitution. The conditions, under which it originates, may be grouped in the following manner:—

1. Idiopathic.	
2. Traumatic	Wounds of pericardium from without, or through œsophagus;[1] foreign bodies from œsophagus;[2] blows and contusions of præcordial region.
3. Perforative	From liver abscess through diaphragm.[3]
4. Attendant on or consecutive to—	
(*a.*) General diseases	Typhoid (Peyerian) fever, variola, scarlatina, pyohæmia.
(*b.*) Diathetic diseases	Rheumatism, Bright's disease, gout, scurvy, purpura, cancerous and tuberculous diatheses.
(*c.*) Diseases of bloodvessels	Phlebitis, endosteal and other, after amputations and various operations.
(*d.*) Peculiar states of the blood	Cyanosis.
(*e.*) Sudden removal of long-established diseases	Psoriasis, chronic eczema, and lepra.
(*f.*) Diseases of pericardium	Cancer, tubercle.
(*g.*) Adjacent diseases	Pneumonia, pleurisy, pneumothorax,[4] inflamed mediastinum, abscess with probably diseased rib,[5] costal periosteitis,[6] abscess under abdominal surface of the diaphragm.[7]
5. Terminal.	

I have never seen idiopathic pericarditis as a solitary disease. But in those rare cases of bilateral pleurisy, occurring in healthy individuals, to which I have elsewhere referred,[8] pericarditis is a usual attendant; and has a claim to be considered idiopathic.

Although indubitable examples of these various modes of origin occasionally occur, they are, all taken together, very rare; and, practically speaking, the complaint is scarcely met with in London but as a local development either of rheumatism, or of Bright's disease.

In rheumatic fever the joints commonly suffer before the pericardium; or the pericarditis may lead the way, the joints following; or both pericardium and joints may suffer simultaneously; or there may be no arthritic affection, pericarditis and acid sweats existing alone.[9] The late Dr. Taylor proved that Bright's disease in its advanced stages acts almost as frequently, as a cause of pericarditis, as does acute rheumatism.

[1] U. C. Museum, No. 3859—Indian juggler's so-called knife swallowed.

[2] Artificial teeth swallowed, lodging above the cardiac orifice of the stomach, and ulcerating through into pericardium.—Amer. Journal, Dental Sc., or Med. Times, May, 1858.

[3] Graves, Clinical Medicine.

[4] Healey, U. C. H., Females, vol. x. p. 91.

[5] Newman, seen with Mr. Quain, U. C. H., Jan., 1854.

[6] Llewellyn, U. C. H., Males, vol. xi. p. 174.

[7] Crawley, U. C. H., Females, vol. x. p. 18, March, 1854.

[8] Diseases of the Lungs, 3d Am. edit., p. 238.

[9] Perry, U. C. H., Females, vol. i. p. 82.

Although pericarditis may by possibility occur as one of the secondary inflammations of typhoid (Peyerian) fever, it must be conceded the tendency thereto is extremely feeble, as M. Louis did not find the least trace of pericardial irritation in fifty-six fatal cases.[1]

Scurvy appears to act in some parts of Northern Europe as a very frequent cause of pericarditis.

The cancerous diathesis very rarely acts as the efficient cause of pericarditis; even cancer of the mamma, so frequent a cause of adjoining pleurisy, rarely excites the pericardium to inflammation.

And the tuberculous diathesis rarely evolves the disease; still I have seen it in phthisical persons (free from real tuberculous disease of the pericardium or heart itself, non-rheumatic, and possessed of healthy kidneys) under two forms. In the one the exudation products are in no wise distinguishable from those of ordinary plastic pericarditis;[2] in the other those products partake of the characters assigned to true tuberculous pericarditis.[3]

Pericarditis, in this respect claiming a distant affinity to pneumonia, sometimes acts as the immediate cause of death in lingering diseases.[4] I have especially known it do so in phthisis.

468. *Physical signs.* (*a.*) During the *dry stage*, the extent of visible impulse is greater than natural; the impulse, as felt, is too forcible, of beating rather than heaving character, and successive impulses are of unequal strength: the action is suggestive of excitement. The areas of percussion-dulness, both superficial and deep [95], are unchanged. Grazing friction-sound may occasionally be caught; and even sharp, though feeble, scraping sound, if there be a prominent tuft of dilated capillary vessels.[5] The physician should, at this period, while it is yet unchanged by the disease, accurately ascertain the point of the apex-beat, to enable him to substantiate its subseqnent displacements.

469. (*b.*) In the *plastic exudation-stage* incipient bulging of the præcordial region may, it is averred by some observers, be pretty constantly detected. In their apprehension "paralysis" of the muscular structure of the intercostal planes, induced by the adjacent inflammation, suffices, aided as it is by the protrusive action of the heart, to bulge the chest-wall. I have never succeeded in discovering this; nor does the theory seem at all sound. Admitting that the inflammation of a serous, in close contact with a muscular membrane, "paralyzes" the latter, as in the alleged instance of pleuritis, it does not follow that pericarditis will have any such effect, seeing that the pericardium is not in contact with the side.

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[1] Affection Typhöide, t. i. p. 328, éd. i.

[2] Ryan, U. C. H., Females, vol. xiii. p. 43.

[3] Mercer, U. C. H., Females, vol. xiii. p. 7.

[4] Healey, U. C. H., Females, vol. x. p. 91.

[5] Parker, U. C. H., Males, vol. iv. p. 177. This is the only case I have ever met with where I succeeded in obtaining the post-mortem demonstration that mere excess of vascularity may generate loud well-defined friction-sound.

The inspection-signs are essentially the same as in the dry stage; but elevation of the apex may be effected by the contraction of lymph solely, if this stage be protracted [54]: the wings of the diaphragm may be somewhat raised too.

The hand sometimes detects pericardial friction fremitus or jerking vibration.[1]

If the plastic exudation be very thick, it is conceivable that the area of superficial dulness shall be extended; but this is a point too delicate to be trusted to.

The essential sign of this stage is pericardial friction-sound, of which the properties have already been described [222].

The condition of the heart's sounds varies; they may be unchanged, or even louder than in health; or, on the contrary, masked somewhat by the loudness of the friction-sound, or even positively enfeebled, especially the first, in all probability by the interference of thick layers of lymph with the full play of the ventricles. I have known the second sound sharply reinforced at the pulmonary, or second left, cartilage.[2] Valvular murmurs are of excessive frequency as dependencies on coexistent endocarditis—especially the aortic constrictive and mitral regurgitant varieties. But may valvular murmur come directly of exudation on the pericardial surface? It is conceivable that the aorta and pulmonary artery may be so pressed on by lymph, that murmur shall be engendered with the systole as the blood passes through the slightly constricted part; but I do not know this from experience. In regard of prognosis, the question is obviously one of importance—a murmur, thus generated, must be of less serious import than one of endocarditic origin. The respiration-sounds continue unchanged over the area occupied by the heart.

470. (*c.*) The perfection of the signs of the *stage of liquid effusion* varies directly as the amount of fluid.

By inspection may be discovered arching of the præcordial region, widening and even bulging of its intercostal spaces, with elevation of the left edge of the sternum, sometimes traceably increasing from day to day, and in extreme cases bulging at the upper and left part of the epigastrium; œdema of the præcordial integuments, especially if the effusion have existed for any time, is occasionally noticeable. Undulating impulse [58], and displacement of the apex-beat upwards as far as the fourth interspace, and slightly outwards, complete the list of physical signs.

By application of the hand, added to auscultation, we find that in cases where the apex lies behind a thick stratum of fluid, the impulse lags slightly behind the systolic sound. The impulse feels weak, unequal, fluttering, or may be imperceptible; and if pericardial friction-fremitus had existed, it is now gone. The line of vocal fremitus at the right side of the heart is carried unnaturally to the right—a valuable sign in some cases. The state of respiration-expansion over the heart varies—if the quantity of fluid be moderate, costal expansion is well marked, diaphragmatic movement being impeded by the fluid—if very great, that expansion is impaired.

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[1] I have never observed fremitus produced by cardiac action on pleuritic lymph, the pericardium being positively healthy; Dr. Stokes, however, appears to have done so. (Op. cit., p. 29.)

[2] Smith, U. C. H., Males, vol. xvi. p. 65, Feb. 19, 1859. But this man eventually left the hospital with a mitral regurgitant murmur.

The interval by measurement between the left nipple and the middle line may be increased; but this is not always the case, even where bulging is well marked to the eye.

Percussion discloses that which is, all things considered, the most trustworthy sign of pericarditic effusion, namely, præcordial dulness of the peculiar triangular outline, the base below, the apex above, which has already been fully described [107]. The area of this dulness may be changed sidewards, most readily to the right, by moving the patient successively from one side to the other.

By auscultation, the irregularity of the impulse in regard of force, and, if this be affected, of rhythm, is better perceived than by other means. The friction-sound of the past stage may be either completely gone; or heard in some spots about the great vessels; or pretty generally retained in the præcordial region—but this is very rare even with eight ounces of fluid, and seems scarcely possible with more than ten. On the other hand, *no conceivable amount of fluid will of necessity totally annul friction-sound.* I base this statement on a case, already referred to a moment since, in which I and others distinctly heard friction-sound "at mid-sternum on the level of the third rib," and yet (death occurring only twenty-nine hours later) sixty measured ounces of fluid were found in the pericardial sac, which reached about a thumb's breadth *above* the clavicle.[1]

The possibility of systolic basic murmur being produced by pressure of fluid on the great vessels has already been referred to [214].

The heart's sounds feeble, distant, and as it were muffled, at the lower part of the cardiac region, become louder as the stethoscope is carried upwards, and at the top of the sternum the second sound is full and loud, and the first very decidedly more marked than directly over the ventricles. This is a sign which occasionally proves of great diagnostic significance.

Various displacements of adjacent structures occur. The anterior edges of the lungs are pushed aside by the accumulating fluid; they may by possibility be separated, on the level of the second cartilages, by a space five inches wide.

Where copious effusion is suddenly poured into both the pericardium and the left pleural sac, it would appear, from a case observed by Dr. Stokes, the left lung may be pushed upwards so as actually to form a prominent elastic quasi-tumor above the clavicle.

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[1] Bartlett, U. C. H., Males, vol. iv. p. 292.

The central tendon of the diaphragm undergoes depression, and may be rendered convex inferiorly; the liver *may* be pushed downwards and to the right, but an enormous amount of fluid will not necessarily displace it.

The respiration-sounds in the centre of the cardiac region are feeble and distant; in some very rare instances the voice resounds with an ægophonic twang at the edge of the effusion; this is especially likely to occur, if the adjacent border of the lung be indurated.

471. (*d.*) In the *stage of absorption of fluid* true undulatory impulse disappears—the sufficing cause of *wavy* movement is no longer present. But a fluttering uneven impulse, by no means wholly dissimilar to the eye, may remain—especially if the thoracic walls be thin. The point of the apex-beat almost invariably falls. The bulging of the cardiac region gives way.

By the hand we ascertain that the impulse has recovered its breadth and fulness; friction-fremitus, too, may return.

The dulness of effusion gradually diminishes from above downwards, and draws in laterally also, but not till it has undergone a very distinct fall superiorly.

Redux friction-sound is caught by the ear, commencing commonly, but by no means always, about the roots of the vessels, and varying in extent with the rapidity of absorption. The churning or continuous rumbling variety is the rarest condition of friction-sound discovered.

The heart's sounds recover their fulness (if endocarditis have not prominently existed), and also their natural nearness to the surface. The præcordial respiration-sounds return slowly, and may never, especially if agglutination of the pleural surfaces occur in front of the pericardium, regain their natural intensity.

472. (*e.*) The occurrence of the last stage, that of *adhesion* of the pericardial surfaces, is announced by disappearance of ordinary friction-sound, and *à fortiori* of friction-fremitus, if this have existed; the former may continue to be distinctly heard in some spots, where adhesion is as yet unestablished. Besides, the clicking variety of friction-sound may long continue audible at the base. The percussion-dulness continues to decrease, or, at all events, does not increase, in area. The action of the heart may be tremulous, unsteady, or jogging, and to the eye distinctly undulatory [59].

473. The moment adhesion is accomplished, the evolution of the acute disease has reached its final term.

474. *Symptoms.* I. *Local.*—The chief local symptom of pericarditis is pain, occupying the cardiac region only, mainly seated in the epigastrium, or extending to the left shoulder and elbow—slight in amount or of agonizing severity, lancinating, tearing, burning, or consisting of a mere sense of soreness; and increased

by movement, and deep inspiration. Pain, however, may be absent, and in the majority of cases is either absent or of slight severity. The varying amount of suffering in different cases is with difficulty accounted for; coexistent phrenic pleurisy seems to explain the acute agony of some patients, but pleurisy *may* be present and the pain moderate, or *vice versâ*. Rheumatism of the diaphragm, phrenic or intercostal neuralgia or neuritis, are all conceivable, but non-proven, causes of excess of suffering.[1] The præcordial interspaces are generally tender. Pressure in the epigastrium causes great distress in some cases; in others it is borne without a murmur. Bichat's overweening faith in the significance of the indications, derivable from such pressure, was one of those errors of observation into which men of the highest genus, when biassed by theory, will occasionally fall.

The action of the heart varies widely in intensity. There may be constant, more or less violent, palpitation, attended with many or few of the grave subjective miseries of that state; or there may be palpitation of irregular or regular rhythm, concerning the existence of which the patient is scarcely conscious; or the heart's beat may not, even to the observer, be appreciably abnormal.

475. II. *General and Constitutional.*—(*a.*) The decumbency is least commonly on the left side, most commonly on the back; the head is generally kept rather high. Orthopnœa, a most inconstant symptom, if present, is not any proof, as has been taught, that liquid effusion has taken place; it may be absent during the effusion period, and first appear after absorption; again, it may occur paroxysmally and irregularly. Far from orthopnœa, or even raised position of the shoulders, being a necessary result of effusion, the patient, where this is most copious, may lie by choice flat on the back, with scarcely any pillow; this was the habitual posture, for days before death, in the case just referred to, where sixty fluid-ounces had accumulated in the sac. In such cases, the least elevation of the head produces a tendency to syncope—and the patient instinctively dreads movement of any kind.[2] The facial expression is generally anxious; the features drawn; in fatal cases, *risus sardonicus* sometimes occurs towards the close. The sleep is fitful and disturbed; and jactitation of the arms, the trunk being kept quiet, not unfrequent in serious cases. Apprehension of death may be a prominent symptom.

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[1] The satisfactory demonstration by Luschka (Schmidt's Jahrbuch, 1853), that the phrenic is not simply a motor nerve, but contains sensory filaments distributed to the pericardium, as well as to the pleura, peritoneum, and abdominal wall, gives a definite character to the hypothesis of phrenic neuritis and neuralgia.

[2] Numerous examples, in addition to that referred to in the text, have fallen under my notice, showing that this is the habitual posture, when the quantity of fluid has passed a certain amount, the precise amount varying probably in different persons. What is the cause? It seems most likely that the posture is assumed simply because the circulation is so obstructed by pressure on the great vessels and heart, that instinctively the patient seeks the aid of gravitation to supply the brain and lungs with blood.

(*b.*) Rigors may announce the invasion of the disease, but they are not commonly severe nor repeated; the skin, subsequently hot as the usual state, may be paroxysmally cold. If perspiration occur, it is not specially acid, unless rheumatism be present—and it *may* be alkaline, even with this combination.[1] Sudamina sometimes form, and their contents may be alkaline, while the surrounding perspiration is acid—or the reaction of both may be the same; none of these conditions are peculiar to the disease. Œdema sometimes occurs, particularly if the case be protracted, about the ankles; and may also appear in the integuments of the cardiac region. The integuments of the head, face, and neck, may become generally livid, with associated swelling of the medium-sized and large subcutaneous veins.

(*c.*) The joints are not affected as a consequence of pericarditis—they are frequently rheumatic, of course, as the majority of cases of pericarditis are of rheumatic origin. The limbs generally are the seat of such pains as appertain to a pyrexial disease.

(*d.*) There is nothing special in the state of the tongue. Deglutition, commonly natural, may be gravely obstructed in the manner to be presently described. I have never met with a case in which pressure of the distended pericardium on the œsophagus even appeared to have acted as an efficient mechanical cause of dysphagia. Tenderness of the epigastrium, with nausea and vomiting of food, or of bile even (the stomach being perfectly healthy),[2] sometimes constitute very prominent symptoms—prominent enough to throw all those of the cardiac class into the shade. The liver grows engorged; I have never seen jaundice. The bowels are confined.

(*e.*) Dry, irritable, abrupt, jerking, spasmodic cough, with variable dyspnœa, and coolness of the expired air towards the fatal close, are the chief pulmonary symptoms. That exaggerated respiration results from pericarditis *per se*, I more than doubt. The voice is sometimes much weakened, an effort required to produce feeble tones:[3] this symptom seems mainly connected with copiousness of effusion, though conceivably it may depend on reflex irritation of the vagus nerve.

(*f.*) The pulse is commonly frequent out of proportion with respiration, unless there be some pulmonary complication. I have, however, known in rheumatic pericarditis, with chorea, a ratio of 136 : 64 or 2.12 : 1, the lungs being free from inflammation.[4] At first full and hard, and sometimes less frequent than natural, afterwards weak and feeble, the pulse grows irregular, both in force and rhythm, in about one-third of cases—or such irregularity may exist from the first, and before fluid is present to account physically for the circumstance—or irregular and uncountable from frequency one day, it may be infrequent and perfectly regular on a subsequent one, the disease meanwhile advancing.[1] The frequency of the pulse is subject to more sudden variations, from the influence of emotional excitement and effort, than in any other disease perhaps: thus gentle movement of the trunk may raise the pulse from 80 or 90 to 130 or 140. The blood is hyperinotic.

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[1] Numerous instances of this exist in my hospital books.

[2] G. Perry, U. C. H., Females, vol. i. p. 82. 1846.

[3] Bartlett, U. C. H., Males, loc. cit.

[4] Leason, U. C. H., Females, ætat. 9, vol. iii. p. 69.

(*g.*) The lymphatic system is unaffected.

(*h.*) Having yet to observe a case of isolated idiopathic pericarditis, I am unable to say what influence the disease *per se* exercises on the composition of the urine. As the complaint is daily seen, in association with diathetic and acute specific diseases, the urine is of highly marked pyrexial type, or possesses the characters of the disease to which the pericardial inflammation is superadded.

(*i.*) I have not met with any special indications of sympathy on the part of the genital organs. Menstruation runs the same, but not a greater, chance of being arrested or deferred by an attack of pericarditis as of other acute inflammations: the function may be wholly unaffected.

(*k, l.*) Slight cephalalgia is common; the pain is rarely sufficiently great to excite spontaneous complaint. As a rule encephalic symptoms of any serious import are totally absent from first to last. But, on the other hand, delirium, apoplectiform stupor with imperfect hemiplegia and a quasi-maniacal state[2] or actual mania of violent character, sometimes give special character to the case. And grave nervo-muscular disturbances, pointing to spinal origin, are occasionally witnessed; among these may be enumerated laryngismus; external cramps; slight clonic spams, scarcely amounting to fits of convulsion; epileptiform, hysteriform, trismal and tetanic seizures, and chorea in all degrees of severity.

These cerebro-spinal symptoms are sometimes in all likelihood produced rather by the altered state of blood coexistant with the pericarditis than by the local conditions of this inflammation in itself: in the pericarditis of Bright's disease uræmia is doubtless their immediate cause. In the rheumatic variety these symptoms may, however, be of reflex mechanism, excited by irritation of the phrenic nerves—a view, though deficient in proof, certainly more tenable, than that ascribing them to cerebro-spinal meningitis: for the positive absence of that inflammation has more than once been demonstrated *post mortem*; and when it actually exists, no one of the symptoms just enumerated may occur.[3] Since I originally noticed dysphagia as a symptom of pericarditis in the first edition of this work, I have seen two additional striking examples of it;[1] in one of the patients, an adult, wildness of manner, tremors, paroxysmal orthopnœa, with faintness and subsultus tendinum coexisted; in the other, a child, violent bilateral choreal movements and imperfect laryngismus. Both cases confirm the opinion formerly expressed, that the symptom is of purely dynamic mechanism.

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[1] Bartlett, U. C. H., Males, vol. iv. pp. 295–6. Rhythm may be regular, while force is irregular.

[2] Campion, U. C. H., Females, loc. cit.

[3] Gash, U. C. H., Males, vol. x. pp. 178–182.

(*m.*) Alterations of the functions of special sense have not fallen under my notice, unless in a few cases where they (blindness and deafness) were plainly referable to the dominant diathetic state present—that of Bright's disease.

476. *Course and Mode of Progress.*—Pericarditis may run a uniform course, with its several periods of increase, status, and decline. Or the disease may at any moment undergo temporary suspension, not only in respect of its subjective symptoms, but of many of its objective conditions. True relapse of pericarditis is certainly uncommon; whereas, as far as I have seen, recurrence is more frequent than in the case of any other inflammation of grave character.

In the great majority of cases the symptoms of pericarditis are sufficiently marked to draw the attention of both patient and observer to the seat of disease; in some few, however, all subjective indications are wanting—the affection, completely latent, is only to be detected by objective evidence. Such latency of course is more common in the diathesis of Bright's disease, than under other circumstances; but I have a considerable number of cases recorded in the hospital books, where, even in rheumatic fever, all the physical signs of exudation and of moderate serous effusion existed without the slightest pain or functional disturbance of the heart. Latency of course may occur under all anatomical conditions of the disease—and seems plainly connected with the special nervous susceptibility of the individual, rather than with the amount of local mischief.

477. *Terminations.*—(1.) When acute pericarditis terminates clinically by *recovery*, what are the anatomical changes effected? John Taylor thought strata of lymph might be completely absorbed; but, formerly acquiescing in this view, I now cannot help contesting it, at least on the evidence adduced by its author. For his evidence simply amounts to this—that, as friction-sound sometimes disappears without signs of adhesion ensuing, lymph, which must have been the cause of that friction, has been absorbed. Now, as it is certain friction-sound may be produced by mere vascularity of the membrane [468], the whole argument falls to the ground. But, be this as it will, the common terminations are by absorption of fluid, with adhesion of the serous surfaces—or without such adhesion, where lymph has been exuded by one of the two surfaces solely.

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[1] Campion, U. C. H., Females, vol. vii. p. 260; and Miss B——, seen with Dr. Neil Arnott.

(2.) Or instead of recovery, in the complete sense, occurring, the acute passes into the chronic disease, either with adhesion and agglutination, and more or less constant tendency to irritative congestion—or with persistent effusion.

(3.) Or death occurs. Commonly syncopal, death may be *gradually* effected either by the mechanical interference of the fluid with the heart's action, or less readily by that of effused lymph. It seems wonderful what an amount of pressure by accumulated fluid can be borne with impunity by the heart. Fifty to sixty ounces do not necessarily stop its contractions; but this obviously depends on the slowness of its accumulation and extensibility of the pericardium; a few ounces of blood suddenly poured out from an aneurism into the serous cavity will, we know, mechanically destroy life in an instant—still the nervous shock of the arterial rupture plays some part in the fatal result. Contraction of effused lymph may so twist and distort the heart, that mechanical suspension of projection of the blood through the orifices becomes well conceivable.[1] Besides, there can be no doubt intrinsic changes in the heart's own muscular condition—dynamic failure and alteration of texture by acute interruption of nutrition—occasionally help to arrest its movements. Or death may *suddenly* take place from raising the head or otherwise moving abruptly—an acute syncope caused by cardiac paralysis. Lastly, the fatal event may be mainly traceable to some one or more of those forms of cerebro-spinal disorder above described.

478. *Duration.*—(*a.*) In rheumatic pericarditis death rarely occurs before the tenth or twelfth day; and may be deferred for upwards of three weeks. On the other hand, in less than thirty-six hours life may be extinct. M. Andral records an instance fatal in twenty-seven hours from the first supervention of pain. The great orator Mirabeau was cut off by pericarditis so rapidly, that the reports of his having been poisoned readily gained credence. (*b.*) In instances of recovery, disappearance of subjective, and of most of the objective, symptoms takes place in from twelve to twenty days; but physical signs may hold on for weeks, nay for months, especially slight percussion-dulness, and clicking friction sound, above the heart's base, and occasionally ordinary friction towards the apex and over the general surface.

479. *Diagnosis.*—The diagnosis of pericarditis can only be made with security by means of the physical signs. For, first, so absolutely *latent* from the beginning to the end may the disease prove, that I have known patients with several ounces of fluid and exudation matter in the pericardium, grow irritated, when inquiries were made about symptoms connected with the heart;[2] secondly, there

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[1] Campion, U. C. H., Females, vol. vi. pp. 40, 41; also Mercer, U. C. H., Females, vol. xiii. p. 4.

[2] This kind of pericardial anæsthesia (if I may use the expression) is particularly prone to occur in patients affected with Bright's disease.

may be a total want of harmony between the violence of the symptoms of which the patient is actually conscious and the amount of disease; or, thirdly, the symptoms of other affections may be simulated.

Before the occurrence of friction-sound there is no certainty in the diagnosis. But if there coexist with sudden excitement of the heart, and absence of endocardial murmur, præcordial distress and tenderness under pressure, while the pulse is frequent (I have not been able to confirm Dr. Graves' observation as to its infrequency), and the respiration non-accelerated, the probabilities pronounce for pericarditis. This will especially be true, if any one of the diathetic or acute specific diseases, with which pericarditis is known to coexist, has preceded.

The essential signs of pericarditis are friction-murmur, special præcordial dulness, and twisting upwards of the heart's apex.

(*a.*) Friction-murmur, when thoroughly developed with the characters assigned it elsewhere, is next to pathognomonic; the possible fallacy from pleural friction of cardiac rhythm must not, however, be forgotten. Mediastinal pseudo-rhonchus (which may coexist, too, be it remembered, with true pericardial friction), is another possible source of fallacy.[1] By a little care the squashy rhonchoid sound, sometimes produced by pressure of the stethoscope over the heart, when the integuments are œdematous, will be distinguished from pericardial rubbing-sound; its rhythm is of course respiratory.

I have nothing to add here, to the rules already laid down for the distinction of endocardial from pericardial murmurs [240]; not very unfrequently troublesome, occasionally impossible, the distinction is nevertheless, in the mass of cases, easily effected.

It must nôt be forgotton that a certain murmur, really intra-pericardial, may be mistaken for other things. The clicking variety of pericardial friction differs, however, from valvular clicks in its non-synchronism with either heart-sound, and in its non-transmission along the aorta and pulmonary artery. Again, exudation may be present, and yet no friction-sound evolved—either because the posterior aspect of the heart only is affected—or because one lamina of the membrane only is coated with lymph—or because recent agglutination has occurred—or because old agglutination prevents attrition—or because serum or pus has been copiously poured out. And to all these causes of the non-occurrence of attrition must be added the mechanical conditions, as they may be called, of the lymph itself; if this be particularly smooth, and slimy to the feel, no friction-murmur will arise.[2]

(*b.*) Next, of the significance of the percussion-results. If the peculiar triangular-shaped dulness be plainly developed under the eye of the physician, and have succeeded to friction-murmur, there is no possible source of fallacy. But if it have not been preceded by friction, the dulness may depend on hydropericardium simply; and if the triangular form have not been produced under observation, there are certain sources of fallacy of a serious character already glanced at [107]. For instance, a weak fatty heart, with quasi-undulatory impulse and feeble sounds, intermittent pulse, and febrile action, may coexist, where the form of the heart's dulness is rendered triangular by the chance presence of old exudation-matter about the great vessels, a tumor, a small solid quiescent aneurismal sac —or even of an excess of natural mediastinal fat. How is the distinction of the cases to be established? Probably, in the whole range of thoracic diagnosis, there does not exist a more difficult problem. This truth is exemplified by the following case. An adult, laboring obviously under acute disease, unable to give any trustworthy account of himself, had all the physical heart-signs mentioned, and in addition orthopnœa and jactitation, while, on his admission to the hospital, no ordinary signs of pneumonia or other thoracic inflammation existed to account for the acute aspect of the disease. I was strongly disposed to regard this as a case of latent pericarditis with effusion, accompanying valvular disease, of which latter affection the signs were obvious; but the impossibility of discovering a shadow of friction-sound, though the patient's posture was varied, the non-elevation of the heart's apex, and the fact that above the third rib the dulness was not so absolute as below it, led me to reject the idea. On *post-mortem* examination (the signs of pneumonia had meanwhile made their appearance—on admission, indeed, the pulse-respiration ratio was 2.7 : 1, and in twenty-four hours had become 2 : 1), the pericardial sac was found free from fluid; but above the base of the heart lay a lump of fat, the simple source of all the difficulty.[1] And it is quite conceivable this difficulty might be increased; for a patch of old exudation-matter near the apex might give friction-sound, while all the other conditions were essentially the same as in the case just referred to.[2]

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[1] Vide Dis. of Lungs, 3d Am. ed. p. 118.

[2] Mercer, U. C. H., Females, vol. xiii. p. 4, January, 1857.

Mediastinal tumor of some size, if it encroach on the cardiac region, may simulate pericardial effusion. But the history of the case; the presence of centripetal pressure-signs, which are never caused by fluid in the pericardium; and the outline of the dulness, which can scarcely by an unlucky chance imitate precisely that of pericardial dulness (a tumor grows in spite of gravity, fluid obeys this), will distinguish the former from the latter. Still the diagnosis must occasionally be profoundly difficult; for Skoda refers to a case, where a trocar was driven into an encephaloid mediastinal mass, under the impression that the instrument was to give issue to pericardial fluid. *Per contra* it may happen that intra-pericardial fluid imprisoned by old adhesions about the great vessels at the base of the heart, and limited to that locality, shall to a certain extent simulate a solid mass in the anterior mediastinum.[1]

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[1] Beckett, U. C. H., Males, vol. v. p. 229.

[2] J. Morris, U. C. H., Males, vol. vii. pp. 154—162.

(*c*.) If the elevation of the heart's apex occur during observation, I believe it to be an infallible sign of pericarditis; simple hydropericardium has never within my experience sufficed to displace that point to any notable amount. But I think it right to warn the student, that, where the change is not actually effected under observation, he must be on his guard against a certain number of sources of fallacy. The chief of these are, as follows: the heart's apex, either as a result of original conformation, or through the long-continued influence of stays, may lie unusually high; it may be raised by the contractile influence of the exudation-matter of a long-past attack of pericarditis; it may be pushed upwards and outwards by an enlarged liver; or upwards and inwards by an enlarged spleen; or it may be drawn aside and upwards by the contraction entailed by old pleurisy; or it may be drawn upwards a space and a half by a contracting excavation in the upper part of the left lung; and, lastly, a slight but real elevation of the heart's apex may take place through retraction of the great vessels and shrinking of the heart's cavities consequent on great hemorrhage. As blowing murmur will exist in the latter case, an erroneous assignment of the position of the apex to the influence of pericarditis is the more likely to occur.

There are, besides, particular combinations of symptoms, occurring in pericarditis, which simulate with considerable closeness those of very different affections. Here is an instance. A female had fever, dry red tongue, extreme epigastric, and no præcordial, tenderness, bilious vomiting and diarrhœa, and perfectly regular pulse; she presented not a single symptom connected with the heart, and was free both from Bright's disease and from acute affection of the joints. How is this state of things, which proved to be really dependent on pericarditis with effusion, to be distinguished from acute gastritis? The physical signs say nothing: friction-sound has already disappeared when the patient is first seen, and a huge stratum of subcutaneous fat with a massive mamma may deprive us of the evidence derivable from percussion.[2]

Again, important though the existence of one of the diatheses or cachexiæ, to which pericarditis is commonly linked, be as an element of its diagnosis, the absence of one or of the other must not negative the positive evidence of physical signs. Some years since it occurred to me to observe a case in which every possible sign of pericardial effusion clearly existed; but I declined to place the diagnosis, "pericarditis," on the hospital bed-ticket, because the patient's constitution was sound, no injury had been inflicted on the chest-wall, and she was free from rheumatism. This was the instance referred to in the table, where an abscess under the central tendon of the diaphragm had been the exciting cause of the pericardial inflammation really present, as proved *post mortem*.[1]

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[1] Richmond, U. C. H., Females, vol. xv. p. 5, Dec. 1858.

[2] Facts, all of them, illustrated by the case of Perry, U. C. H., Females, vol. i. p. 82.

In the diagnosis of a difficult case, the functional and general symptoms should not be forgotten, but clinical experience compels us to admit that they are sometimes utterly fallacious aids. Hope wrote the singular proposition, that "the variability of the symptoms is calculated rather to enlighten than perplex the practitioner," his belief being that the symptoms of lymph-deposit were slight, of effusion serious. The value of this dogma appears from a single case, the fourth in Andral's Collection, where death occurred from plastic pericarditis in twenty-seven hours, without a drop of fluid having formed in the sac; while *per contra* the disease may be latent with extensive hydro-pericarditis.

480. *Mortality and Prognosis.*—There are no existing documents from which the absolute mortality, or the ratio of deaths to seizures, may be safely calculated on a large scale.

The *immediate* prognosis is in the majority of cases not unfavorable. M. Louis long since estimated the deaths at one in six of those attacked. But the result is greatly under the influence of the existing diathesis. I do not remember to have seen rheumatic pericarditis fatal, in a previously healthy person, except in some seven or eight instance; whereas a large proportion of cases of pericarditis, arising in Bright's disease, end fatally. Dr. Taylor found in seventeen cases of recovery the mean age = 19.7 years; in twenty-one cases of death = 35.5 years; but renal disease would be more likely to exist in the series of more advanced age than in the other; age alone cannot, therefore, be charged with the difference in mortality. The whole class of reflex phenomena are of excessively bad augury; when highly marked choreal symptoms have occurred in childhood, I have never known recovery ensue. Dysphagia at any age is of very fatal significance.

In regard of *remote* prognosis: if mere adhesions or even agglutination exist, the health does not necessarily suffer; if hypertrophy, simple or dilated, or atrophy, supervene, symptoms of course arise, M. Beau found in forty-eight cases of agglutinated pericardium forty examples of dilated hypertrophy, but the relative dates of the two conditions cannot always be established from his facts. I have seen no reason to accept the doctrine that old pericarditis in itself kills: coexistent endocarditis with its valvular sequelæ is the real destroyer in chronic fatal cases.

481. *Treatment.*—In its character of an acute inflammation, affecting sometimes directly, always indirectly, the dynamic and the statical conditions of the heart itself, simple pericarditis theoretically calls for antiphlogistic treatment of a most active kind. But

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[1] Crawley, U. C. H., Females, vol. x. p. 18, March, 1854.

as matter of actual experience we find that, while isolated idiopathic pericarditis scarcely ever exists, the form of diathetic state, on which the disease supervenes, forces us very materially to modify any therapeutic system, founded on the mere idea of phlogosis.

482. In order to avoid hyper-division, we may consider pericarditis *quoad* treatment, as referable to two varieties: a variety belonging to rheumatic fever, and a variety in which the inflammation appears as a local expression of some established cachexia. The latter variety may be considered to be typified by the pericarditis of Bright's disease.

Now the absolute necessity of distinguishing these two varieties flows from the fact, that rheumatic pericarditis in the great majority of cases terminates favorably, no matter what the precise mode of scientific treatment adopted; whereas actively acute pericarditis of renal origin almost as invariably proves fatal.

483. (1.) *Rheumatic pericarditis.*—(*a.*) *Sthenic.* Whether the pericarditis appear after, along with, or before the articular affections, possibly the intensity of the pericardial inflammation might be lessened by artificially irritating the joints. To grant this, obviously does not require one to have any faith in the exploded doctrine of metastasis; but the *practice* has disappeared, perhaps unmeritedly, since the *theory* has been banished from the schools.

484. Bloodletting claims consideration foremost among professed remedial agents in rheumatic pericarditis. Now, in the first place, the following truths show the wisdom of caution in the amount of blood abstracted. Bleeding from the arm cannot be pushed to any notable extent without the risk of inducing syncope—an occurrence of serious danger when already the heart's vigor is dynamically and statically impaired. Venesection, be it ever so free, not only does not *of necessity* arrest, or very sensibly modify the course of the existent pericarditis—but may actually fail to prevent the development of other inflammations (for instance, pneumonia), secondary to itself. Very copious depletion, particularly in some constitutions, excites the heart greatly, and in several respects makes matters worse than they were. And, further, the most severe rheumatic pericarditis may go on equably or interruptedly to recovery, though very moderate cupping or leeching over the præcordial region have been substituted for profuse phlebotomy. Yet, on the other hand, I must candidly own that to my apprehension the query whether bleeding at the very outset may ever succeed in arresting all phlogistic action at once, has never yet been scientifically answered in the affirmative or the negative. Instances in which the inflammation is positively caught in the nascent state are rare; and if treatment appeared to have attained its object, the accuracy of the diagnosis at so early a period of the disease might reasonably be questioned. Meanwhile it is certain, lavish depletion in some cases lessens pyrexia and calms local pain and distress, not only without preventing, but without a jot abating

the activity of, the exudation and effusion-processes. Meanwhile, too, that the directly curative importance of copious bloodletting has been exaggerated, while its special dangers were ignored, no reasonable doubt can be entertained. In the fulness of his enthusiasm, Dr. Hope wrote, "the loss of a few hours at first may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. Venesection to the verge of syncope, followed by twenty-five to forty leeches," he holds to be essential in severe recent cases; and "unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be *repeated two, three, or more times*." What then is to be said of a case, which I have elsewhere published, where violent acute rheumatism coexisted with pericarditis, endocarditis, double pneumonia and pleuritic effusion, and probably aortitis, and yet, twenty-two ounces only of blood having been taken by cupping from the cardiac region, convalescence was established by the twenty-first day?[1] I never draw blood largely from the arm in rheumatic pericarditis; and, as I formerly said, "the reason why I abstain from the practice are not that I fear people profusely bled shall 'forthwith go raving mad' (as, according to the statement of Dr. Latham, they nevertheless have, though in rare instances, actually done); nor that I fear endocarditis shall forthwith be generated —an effect which has by some authors been positively assigned to the practice, but which effect the said practice has much more certainly never been proved to entail. My reasons are, first, that none of the advocates of such venesection have ever shown that the mortality, duration, or suffering, of the disease, are less in a series of cases thus treated, than in a series of cases treated by gentler means—by colchicum, alkalies, and mercury, for instance. And secondly, that while, by prodigal bleeding, a loss of vital fluid is inflicted, which it may take the nutritive system months, or even years in individuals of certain constitutions, to repair, all that loss may be saved without demonstrated risk of any kind."

On the other hand, that moderate venesection shortens the duration of pericarditis, and does so more effectually the earlier it is performed, has been clearly shown by Dr. Taylor in his logical papers on the treatment of the disease. The quantity of blood to be drawn must be regulated by the severity of the symptoms: from an adult of medium strength some eight to twelve ounces may be taken from a vein in the arm, the head being kept *low*, especially if there be much fluid in the sac. This depletion may be followed, if well supported, by the abstraction of some six or eight ounces more by cupping or leeching over the heart.

I have known moderate bloodletting improve, within a few hours, the tone of the first sound, probably by exciting absorption of fluid in the sac, and facilitating cardiac action.

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[1] Clinical Lectures, case of Craddock, Lancet, February, 1849. I have repeatedly seen this man since 1849 with relative slight attacks of rheumatism. In the intervals of these seizures his health and muscular vigor are perfect—and such as I do not imagine he could have regained had he been lavishly bled.

In cases of medium and of slight severity, venesection may, with advantage, be replaced by the application of a few leeches to the præcordial region, or by the removal of blood to the extent of four or six ounces by cupping. And, as far as my own experience of late years is concerned, it would appear that cases of medium and slight severity must, to my apprehension, greatly outnumber those of graver character; for a considerable time has passed since, as far as I remember, I have employed any more active depletory measure than the application of some eight to twelve leeches.

485. Although much has been more or less eloquently written on the powers of mercury in rheumatic pericarditis, especially on the faculty it possesses of causing the absorption of, and repairing the damages effected by, exudation-matter, it must, I think, be conceded, that any precise evidence before the profession fails to demonstrate the alleged prowess of the mineral. Ptyalism is with difficulty induced; and when induced does not, of necessity, arrest the exudation or effusion-processes. Dr. Taylor notes that in three of his cases (no very small proportion of the whole number) increase of signs and symptoms manifestly followed salivation; and, it is inferrible, he observed many others wherein such increase, though less in amount, was not less real as matter of fact. Dr. Taylor's data indeed, taken as a whole, distinctly argue against the efficacy of mercury. He refers to the frequent occurrence of sundry acute inflammations during the progress of salivation, induced for the intended cure of others. For my own part, I have known pericarditis supervene in a woman while under treatment for ptyalism, so severe, that for some hours after her admission into hospital for the cure of that ptyalism, life was in danger from semi-asphyxia, and tracheotomy on the point of being performed.[1] Mercury is supposed to act in inflammations by diminishing the quantity of fibrin, yet M. Andral found hyperinosis in four cases of mercurial stomatitis.[2]

I must admit that year by year my faith in mercury has waxed less strong—and that a long period has elapsed since I have made even a deliberate attempt to salivate a pericarditic patient.

486. On the principle of regarding the disease through its diathesis, alkalies are advisable, where the pericarditis is rheumatic; I formerly thought colchicum should never be omitted in a case of this kind, but of late years I confess my belief in the real efficacy of this agent in rheumatism has greatly lessened. In overdoses it may induce sudden and dangerous failure of the heart's activity. Opium becomes a necessary remedy in full doses, should agitation and disquietude be at all marked; morphia may be used endermi-

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[1] Spratt, U. C. H., Females, vol. iv. p. 471.

[2] Hématologie, p. 90.

cally, if it has been found advisable to apply a blister to the præcordial region. The application of a blister there is, however, objectionable from its interfering with the examination of the cardiac region, and hence preventing a precise knowledge of the state of the disease; blisters should rather be applied behind, than actually on the præcordial region. Digitalis, aconite, and hydrocyanic acid are dangerous agents, from the chance of their increasing tendency to syncope. Purgatives, diuretics, and diaphoretics are advisable as aids in the treatment.

487. Sinapisms, frequently repeated, are of great service in relieving pain and distress, and are not open to the objection just mentioned in the case of blisters. Ioduretted frictions, coupled with mercury in very small proportion, seem to promote absorption of exudation-matter, especially when the more acute state has passed by.

The hot-air or vapor bath is a valuable adjuvant—both have the advantage over the warm bath, that they may be taken with the head moderately low.

488. The regimen must be essentially antiphlogistic; as a matter of no small importance, the patient should be strictly cautioned, if there be much fluid in the sac, against abrupt movements and elevation of the head. In cases where failure of the heart's force occurs, whether early or late in the attack, animal food, diffusible stimulants and brandy are called for. Even though this failure come of actual inflammation of the heart's fibre, the pulse must be sustained by alcoholic stimulus.

489. No special treatment is required for the various inflammations sometimes occurring secondarily. But the whole class of reflex symptoms call for the suspension of directly lowering measures and of mercury; purgatives, antispasmodic and sedative remedies must at once be had recourse to; and an attempt made to rapidly excite irritation in the joints by sinapisms. If dysphagia arise, the neck should be blistered.[1]

490. (*b.*) *Asthenic.*—In asthenic rheumatism, no matter from what sources the asthenia springs, pericarditis should not be treated antiphlogistically. Carbonate of potash and of ammonia internally, counter irritation, good nourishment, and moderate doses of wine or brandy, form the staple agents of treatment. If the notions of the late Dr. R. B. Todd were accepted, the administration of brandy should be conducted with a lavish hand, not only in the present, but even, in the markedly sthenic, variety of the disease. But to my mind his argument fails to carry conviction with it; and the fact that a young female, laboring under violent pericarditis, may absorb a bottle of brandy *per diem*, and recover, seems to me simply

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[1] In a recent essay (Clin. Notes on Pericarditis) Dr. Gairdner advocates a system of treatment which seems closely allied to "pure expectation;" and he "has not had under his care a case of rheumatic pericarditis fatal during the acute period of the attack."

to show how difficult it may prove to overcome the tenacity of life with which Nature in rare instances endows us.

491. It seems not unwise to suggest for the thoughtful consideration of those who now occupy the benches of the schools, and many of whom are disposed, with the enthusiasm incident to youth, to become the future practical exponents either of the extreme antiphlogistic, or of the extreme stimulant doctrine, that exaggerated courses of procedure in dealing with complicated and unintelligible derangements of vital actions, are, on logical grounds, gravely wrong. Neither he who pours away the blood of his patient and dreads stimuli as deathly, nor he who looks upon the sacrifice of an ounce of blood as seriously detrimental, and reverences brandy as alone restorative, knows even in a fragmentary and imperfect manner the immediate and intimate effects of the methods he employs. On the ground then that a given cause will produce a certain given effect, and none other, these extreme measures are indefensible, especially as the unknown portion of the effects, where the cause is so powerful, may, for aught that is proved to the contrary, be disastrous in the extreme. Suppose, however, that one of these ultra-therapeutists, professing total indifference to the nature of all immediate and intimate vital changes induced by his method, points exultingly to his results, and demands to be judged by these; the other forthwith proves the vanity of the exultation by proclaiming the success of his own. The two groups of results, in their quality of evidence supporting one or other method of treatment, obviously destroy each other. And so the fancy intrudes itself, that perchance in both series Nature has been the real victor over both disease and treatment.

492. (2.) *Pericarditis of Bright's Disease.*—Bleeding to any extent in the pericarditis of Bright's disease, occurring as it mainly does in the advanced periods of that affection, when the blood is deeply tainted and the nervous vigor impaired, is at once theoretically contra-indicated, and as matter of experience has been shown to be injurious. The activity of the congestive and exudative processes may, however, be somewhat controlled by the abstraction of a few ounces of blood from the cardiac region by bleeding or cupping. Counter-irritation by blisters, applied in the neighborhood of the heart, is also advisable.

General opinion seems to me to have wisely decided that the administration of mercury is, on the whole, injurious in all diseases attended with albuminuria—and especially in the group of diathetic affections included under the head of Bright's disease. Iodide of potassium and bicarbonate of potash, in full watery solution, are probably the medicines best calculated to act diuretically in removing the uræmic poison from the blood, which may fairly be regarded as the exciting cause of the pericarditis. The hot air, or the vapor bath, and hydragogue purgatives will favor elimination by other channels. Dry cupping over the kidneys, by lessening congestion, promotes their secretive activity.

B.—CHRONIC PERICARDITIS.

493. Under the head, chronic pericarditis, may be included four states clinically very different; namely (*a*), that in which adhesions or agglutinations of the pericardium having formed, a tendency to active congestion in the pericardium itself, and in the substance of those adhesions, is more or less constantly present;[1] (*b*), that in which cardiac hypertrophy is conjoined with such adhesions; (*c*), that in which cardiac atrophy coexists with such adhesions; (*d*), and that in which liquid effusion remains in the sac without apparent inclination to increase or to disappear.

(*a*.) SIMPLE PERICARDIAL ADHESION.

494. In the first form, that of adhesion, there may be, in the absence of irritation, a complete nullity of symptoms, neither in cardiac action subjectively considered, nor in the state of feeling about the heart generally, is there anything to arrest attention. If there be congestive tendency, palpitation is easily excited, the breath short, and uneasiness of various kinds readily induced.

495. We have seen what the physical signs of adhesions are at the time of formation; enlightened, as the observer then is, by the previous course of events, they are easily established. But in a case, seen for the first time, after adhesions have been some while formed, their positive diagnosis is among the most difficult clinical problems existing. Nay, more, I believe the achievement an impossible one in a goodly proportion of cases; I have known adhesions found after death where, despite frequent physical examination of the heart, their existence had not been even suspected; and, *vice versâ*, seen the pericardium of model health, where an adherent condition of the two laminæ had with overweening confidence been affirmed. Still, though difficult, the attempt must not be given up—and the following points will help the observer materially.

The heart itself being either quite healthy, or not seriously changed in bulk or structure, the signs will vary, first, with the closeness of the pericardial adhesions themselves, and secondly, with the absence or presence of pleuritic adhesions in front of the heart. So essential is the latter point, though ignored by writers generally on the subject, that it forms the ground of a distinction of the physical signs into two classes, as follows:—

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[1] Vessels in old adhesions will sometimes bleed freely on being torn across, *e. g.*, Mann, U. C. H., Females, vol. ii.

Physical Signs of Pericardial Adhesion.

(*a*.) *Pleuritic adhesions in front of the heart absent.*	(*b*.) *Pleuritic adhesions in front of the heart present.*
Apex-beat in natural site; or retained above, in the spot it was raised to during the acute stages.	The same.
Apex-beat not notably lowered by inspiration.	This peculiarity still more marked.
Sometimes distinctly defined apex-beat wanting.	The same.
Depression of præcordial region, about fifth to seventh left cartilages; or superiorly at the base of the heart.[1]	This more marked.
No dimpling of surface with cardiac action.	Epigastrium at left costal angle may dimple inwards with the ventricular systole.
No undulating movement of heart.	Undulating movement more or less, sometimes excessively, well marked.
Respiration-movements slightly less active over heart than natural.	Respiration-movements almost null over heart.
Percussion-dulness about large vessels above third left cartilage from pericardial induration-matter.	This still more marked from the additional influence of pleural induration-matter, and condensation of neighboring lung-substance.
Pericardial clicking sounds about roots of vessels, or common friction-sound from loose adhesions,[2] or slight single rub with systole.[3]	The same.
Complete agglutination may exist without jogging, trembling motion of the heart.	Jogging trembling motion of the heart: (still, even here, this is rare, if the heart's bulk be natural.)
Even though the two laminæ be agglutinated, the heart will fall from side to side as the patient's posture is similarly changed from right to left.	With such additional adhesions the heart will be fixed, and not movable from side to side.

It has been said that feebleness, even to extinction of the second sound, is a sign of pericardial adhesion; I doubt whether the two things, when associated, have ever any direct connection; and I know that complete agglutination may coexist with a perfect second sound, and this, I may add, even though the wall of the heart be locally infiltrated with cancer.[4]

If new effusion occur in a formerly inflamed pericardium, the continuance of respiration in the præcordial region is a sign of adhesion, pericardial and adjacent pleuritic combined.[5] A past pericarditis does not prevent the characteristic signs of effusion occurring a second or a third time, provided there be not complete agglutination; friction-sound, percussion-dulness of triangular outline, and twisting of the heart's apex upwards may all be present with, and through, the new attack.

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[1] Case of Craddock, Clin. Lectures, Lancet, loc. cit.

[2] Kennedy, U. C. H., Males, vol. i. p. 67 [470].

[3] Jones, U. C. H., Males, Feb. 1861; proved to have depended on an annular belt of calcification in the agglutinated laminæ; heart in U. C. Museum.

[4] Scammel, U. C. H., Males, vol. xvii. p. 276.

[5] Craddock's case, Lancet, loc. cit. 1849.

(*b.*) AGGLUTINATION OF PERICARDIUM COMBINED WITH CARDIAC HYPERTROPHY.

496. In the second form, where agglutination of the serous surfaces and hypertrophy of the heart coexist, each condition modifies the signs of the other. Thus the heart's apex may beat as high as natural in spite of the enlargement; if, however, the hypertrophy be very great, the apex falls, for the material of agglutination stretches. An unusual tendency to extension of percussion-dulness upwards is observed. Systolic dimpling is very marked; the lower end of the sternum and the adjacent left cartilages may be drawn in at the left costal angle. Jogging, trembling action of the heart may be highly marked.

(*c.*) AGGLUTINATION OF THE PERICARDIUM COMBINED WITH CARDIAC ATROPHY.

497. The cardiac atrophy, distinctive of the third form, comes of tight embrace by the material of adhesions; especially, in all probability, if the coronary arteries be accidentally pressed upon. But such atrophy is of excessively rare occurrence: a fact I ascribe to the rarity of thick strata of true induration-matter in chronic pericarditis.

498. The signs of simple adhesions will be conjoined with those of diminished size of the heart.

499. *Treatment.*—In the three varieties of condition, just reviewed, the treatment in regard of the existing material of pericardial adhesion is identical. The absorption of that material may be encouraged by the use of ioduretted applications, by mercurial inunction, and by the internal administration of liquor potassæ and iodide and bromide of potassium. A course of the Woodhall or Kreuznach waters, internally and externally, is always worthy of trial.

If irritative action supervene, local depletion, blisters, caustic solution of iodine, with small quantities of mercury and digitalis or aconite internally, and free purgation, are the chief remedies. Subsequently the external use of belladonna and aconite will be found useful.

When dilated hypertrophy supervenes, the treatment must be essentially directed to that affection, on the principles elsewhere explained. And, certainly, the chief evil to contend with is generally hypertrophy and dilatation of the ventricles, mainly the left: where agglutination exists, this mode of enlargement of the heart almost invariably commences within a short period. But I confess that my observation does not lead me to take the very gloomy view expressed by some writers of the ultimate issue of such cases: I have not, as they appear to have done, seen mere chronic adhesive pericarditis, with hypertrophy, prove rapidly fatal. The question, becomes, of course, a very different one, if there be valvular disease superadded.

(*d.*) CHRONIC PERICARDITIC EFFUSION.

500. The symptoms and signs of fluid stagnating, chronically, in the pericardial sac are very closely those of the effusion-period of the acute disease.

501. If these signs continue unchanged by ordinary means of treatment (hydragogue purgatives have little effect), paracentesis of the pericardium becomes justifiable, as an *ultima spes*, provided urgent suffocative symptoms exist. The patient is certainly not placed in a worse position by the operation, than he was before it; the immediate relief is extreme, and a certain very small chance exists of at least temporary recovery. Many years ago Professor Romero, of Huesca, published the results of eight cases, three without, five with, coexisting hydrothorax: in two of the former three, the operation was successful.

If paracentesis be determined on (the danger of wounding the mammary arteries, the larger pericardial vessels, and the great arterial trunks being borne constantly in mind), the integuments should be incised opposite the central part of the heart in the natural state of things, that is, at the upper angle of the fourth left interspace [11], or a little lower than this; a trocar should then be introduced cautiously into the distended sac perpendicularly to the surface, the patient being in the recumbent position with the head moderately low.

The fluid, which escapes by jets, corresponding to the ventricular systoles, should be evacuated as completely as possible before the wound is dressed, a syringe may even be employed to insure this. But the orifice of the canula, before its removal, should occasionally be closed, lest too rapid abstraction of the fluid might produce evils of its own on the heart, accustomed as this has been, for a greater or less time, to considerable pressure. The practice of Romero, of allowing the fluid to gravitate into the pleural sac first, and thence outwards, seems unworthy of imitation.

M. Aran records a very remarkable case, in which he twice, within twelve days, tapped the pericardium, giving outlet on the first occasion to twenty-eight, and on the second to forty-nine, ounces of fluid. After the fluid had escaped, the operator on each occasion injected an ioduretted solution into the sac, on the plan which has sometimes proved curative of pleuritic effusion. As far as the operations were immediately concerned, the result seems to have been satisfactory.[1]

Some further information on this subject will be found under the head Hæmopericardium.

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[1] Med. Times and Gaz., 1856.

C.—WHITE PATCHES OF THE PERICARDIUM.

502. A few words may here be added, as a sort of appendix to the subject of chronic pericarditis, concerning the so-called white patches of the pericardium.

503. These patches are of two kinds, perfectly distinct in their anatomical characters and in their mode of origin.

504. (*a.*) In one variety these patches present themselves indiscriminately at all parts of the cardiac pericardium. Transparent at first, they eventually become opaque, white and tough, have well-defined margins, and are more or less readily separable from the serous membrane, which presents the appearances of health beneath them. They are evidently the result of an exudation-process on the pericardial surface, inflammatory in nature in the majority of cases; oftentimes acknowledged inflammatory conditions appear close by.

505. (*b.*) In the other variety, the patch, scarcely ever met with except on the anterior surface of the heart, and principally of the right ventricle, has no sharp, well-defined margin, but is gradually lost in the surrounding pericardium, from which it cannot be separated by any manipulation. There is here a thickening of the proper substance of the serous membrane. The probability seems to be that they arise from long-continued friction, like corns from pressure, of the anterior surface of the heart.

506. The separable patch is rare, the non-separable common. Bizot found the latter variety in 45 of 156 bodies. The same conscientious observer ascertained that this patch is of about three times more frequent occurrence in the male, than the female; and also that its appearance is notably under the influence of age; it is scarcely ever, if ever, seen before the age of seventeen, and does not become common until after forty.[1]

507. Little practical importance can be attached to either variety. If, however, the patch be at all prominent, and especially if it be rough on the surface, it may give rise to single systolic rub of maximum intensity in the lower sternal region.

II.—ENDOCARDITIS.

508. Endocarditis, like the inflammation of the outer membrane of the heart, occurs in the acute and chronic forms.

A.—ACUTE ENDOCARDITIS.

509. *Anatomical characters. In the membrane itself.*—Here redness, florid in tint, sometimes distinctly vascular to the eye (depending, according to Luschka, on injection of the vessels in the deep elastic layer and connective tissue of the endocardium), may exist, pretty

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[1] Mém. de la Soc. Méd. d'Observation, t. i.

generally in patches, or sometimes more uniformly; or the hue may be pale dingy grayish yellow. The surface, deprived of its natural polish, feels more or less rough; the membrane becomes opaque, probably from filtration of exudation through the epithelium, grows thick, soft, velvety, and can be stripped or scraped off in short patches; its tissue sometimes cracks, fibrinous coagula forming in the resulting fissures. *On the free surface.*—In this position particles of exudation-matter, at first gelatiniform and transparent, subsequently opalescent, form thin strata which may be peeled off, or small oval elevations, the latter either on the general surface or on that of the valves or amid the tendinous cords. *Under the endocardium* sero-fibrinous exudation-matter is deposited in small quantities.

Accidental characters are rupture of the tendinous cords, especially of the mitral valve, which then curl up and act as the nuclei of fibrin coagulation: fissuring of the semilunar valves, with deposits of exudation and of fibrin directly from the blood; altered shape of the valves; and softening with rapid destruction of their substance.

Ulceration, or at least destructive softening of the endocardium, may be followed by extension of the process beneath; an opening has thus been effected between the auricles. Pus sometimes forms in the muscular tissue, immediately beneath the endocardium; upon its free surface, in fissures; and, it is alleged, in the substance of fibrinous coagula, softened fibrin has in the latter position, in some instances at least, been taken for pus.

The formation of large coagula within the heart is frequent in severe cases: they bear all the acknowledged marks of production previously to death (the moment of their appearance can sometimes be fixed by physical signs during life), are sometimes the seat of recent vascularization, and are commonly softened in the central parts.

The endocardium of the left side is much more frequently inflamed than that of the right; the inflammation may, however, be limited to the right; affect both sides; or mere fractions of one. The valvular apparatus is more commonly and more strikingly involved than the general tract of the membrane. Endocarditis of the right side in the fœtus has been suggested as a probable cause of coarctation of the pulmonary orifice, and hence of non-closure of the foramen ovale.

Dr. John Taylor was the first to show, as matter of observation, that the products of endocarditis, washed away with the blood, might lay the groundwork of pulmonary, hepatic, splenic, and renal secondary inflammations.

510. *Physical signs.*—In acute inflammation of the endocardium, the heart's movement is seen and felt to influence the surface more extensively, more forcibly, and more abruptly than natural. The cardiac region is not bulged, nor is the point of the apex-beat, as

in pericardial effusion, raised upwards; it may be even carried a little downwards and outwards. There is no undulatory movement, and no tactile thrill; Hope says he has observed the thrill of mitral regurgitation, but he gives no proof that the regurgitation was purely recent; I have not succeeded in finding thrill, when any certainty existed of the absence of old-standing mitral disease.

The areæ of the heart's dulness, both superficial and deep-seated, undergo trifling increase; the former because greater energy of action brings the heart more uniformly forwards; the latter because the walls of the organ are turgid, and its cavities more or less clogged with blood. The area of dulness is never seriously increased, unless there be considerable distension of the heart by accumulated blood.

Auscultation discovers a murmur or murmurs, blowing in quality, soft and low-pitched. The murmurs of *purely acute* endocarditis may, as far as I have observed, be thus arranged in order of frequency; aortic obstructive; mitral regurgitant; aortic regurgitant; aortic obstructive and mitral regurgitant together; aortic obstructive and regurgitant together. Pulmonary systolic and diastolic murmurs are infinitely rare. I have never observed *acute* obstructive mitral murmur, nor *acute* regurgitant tricuspid murmur; the latter, especially, I believe to be at the least very rare, a circumstance in accordance with the fact that most chronic tricuspid regurgitant murmurs are produced by simple incapacity of the valve to fill the widened orifice, without actual disease of its own tissue.

The site and rhythm of acute endocarditic murmurs, it is supposed, may change during the course of an attack; lymph, it is presumed, may be absorbed, or washed away and deposited elsewhere, and a different species of murmur be consequently developed. I have not observed this; but I have known systolic aortic followed by diastolic aortic murmur, apparently from lesion of the valvular structure.

The mechanism of the murmurs of acute endocarditis varies somewhat with the orifice concerned. But surface-roughness, fissuring, lymph-deposits are the main causes of the obstructive class of murmurs; while those of regurgitant character may be traced to intertwined lymph interfering with the play of the tendinous cords, columnæ carneæ, and papillary muscles—to actual destruction of the substance of valves—or possibly in the instance of the aortic valves [207], and probably in that of the mitral [205], to a disordered dynamic condition of the apparatus concerned in closure. It is further conceivable, that friction of the blood over the general surface of the ventricular endocardium, roughened by lymph, may prove the efficient cause of murmur.

Such heart-sounds, as are not replaced by murmurs, present no constant character. Reduplication of the second at the base is common. Probably, at the outset, they are both intensified; and murmur-like prolongation of the first sound, before this actually gives place to true murmur, is sometimes noticeable both at the base and apex.

In cases where the circulation through the heart's cavities is obstructed seriously, either from accumulation of lymph and fibrinous coagula, or from rupture of a valve or chorda tendinea, the impulse becomes irregular in force and rhythm—at first violent, subsequently feeble; the heart's dulness notably extends, especially to the right of the sternum; the sounds or pre-existing murmurs are enfeebled, or new murmurs may be generated. The suddenness of occurrence of these signs points to their source.

511. The *local symptoms* of endocarditis are not very marked. Pain is rare, discomfort and uneasiness at the heart common; more or less palpitation exists; tenderness of the præcordial interspaces is at the least unusual, unless there be coexistent pericarditis [318].

512. The *general symptoms* vary with (1) the free, or (2) obstructed state of the circulation through the heart.

513. (1.) In the first, or free, class of cases the decumbency is generally dorsal, the attitude quiet; but jactitation of the arms occurs in some instances. The skin of febrile heat, the integuments unchanged in color; the joints rheumatic, or unaffected; no special sensation of dyspnœa is complained of; the respiration holds its natural ratio to the pulse, so long as the orifices are not seriously obstructed, and no secondary pneumonia has occurred; sometimes a little dry cough exists without bronchial or other rhonchi; the pulse is not remarkably accelerated, ranging between 80 and 120—Dr. Taylor's statement that it loses in frequency at the outset of endocarditis, I have not had an opportunity of confirming. The blood is hyperinotic in the sthenic disease: if the inflammation be the effect of phlebitis, pyohæmia, &c., the clot is soft, but little or at all buffed: endocarditis may also, in all probability, secondarily, cause the latter condition of the blood by the circulation of its own inflammation-products. The urine is simply febrile. Cephalalgia exists commonly more or less—slight wandering may occur at night, but otherwise the head remains free. I once saw acute mania occur during the convalescence of endocarditis, of rheumatic origin, and unaccompanied with pericarditis.[1] Choreal, or other reflex, symptoms are not induced, if the disease remain simple.

514. (2.) In the *obstructive* class of cases, the action of the heart, suddenly at the moment of obstruction, becomes excessively frequent, uneven, and irregular; the pulse small, weak, irregular in force and rhythm, mounts to 130, 140, 160, or even more. Semi-syncope, pallor, coldness of surface, anxiety, and jactitation, inclination to orthopnœa (which the patient resists from its increasing faintness), with, eventually, the symptoms of more or less complete pulmonary obstruction, lividity of surface, turgescence of the face and neck, prominence of the eyeballs, puffiness of the ankles, super-

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[1] Cooper, U. C. H., Males, vol. i. p. 129.

vene—the brain suffers also congestively, as exhibited by fitful snatches of sleep, convulsions, delirium, and somnolence, lapsing into fatal coma. I have seen these symptoms in a minor degree, and passingly, in certain cases of endocarditis, which terminated favorably, in all probability, in those instances, small concretions had formed, and subsequently undergone disintegration and solution. The symptoms of rupture of a chorda tendinea during the acute disease, are extremely similar; the effects on the cardiac circulation must, indeed, be closely analogous.

The blood, in certain cases of endocarditis, receiving the products of the inflammation, undergoes alteration of composition. Rigors, heat of surface, and profuse perspiration recurring irregularly, dull, earthy, yellow discoloration of the skin, but not of the conjunctivæ, diarrhœa, more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, subsequently cool clammy skin, and blueness of the finger-ends, announce this occurrence; and are followed by, or associated with, the evidences of secondary nodular pneumonia or hepatitis.

515. Rheumatic endocarditis may run a perfectly *latent* course in regard of local and general subjective symptoms.

516. *Causes and conditions of origin.*—Coarising with endocarditis, or generated at least by the same influences, may be found rheumatic arthritis, pericarditis, carditis, aortitis, pneumonia, and pleurisy. Aortitis is a much rarer concomitant, at least as far as proof goes, than might be expected. Bright's disease, rheumatism, and pyohæmia, are the only diathetic affections, that clearly promote the occurrence of endocarditis. Neighboring inflammations, old cardiac disease, injuries to the præcordial region, and violent efforts are sometimes traceable as its causes. The existence of old valvular disease renders the endocardium more prone to acute inflammation.

517. *Diagnosis.*—The diagnosis of acute endocarditis is essentially based on the existence of febrile action, cardiac uneasiness, excited action of the heart, and endocardial murmur—this murmur occurring in a person presumedly free from prior endocardial disease, and presenting no other obvious acute affection to explain the pyrexia.

But suppose an endocardial murmur is detected on the first examination of a patient, laboring obviously under some acute febrile attack, and concerning whose past cardiac history no trustworthy information is to be had, that murmur may obviously be *new*, or it may be *old*. Now, granting that it is new, it may not be the product of endocarditis. For the excitement of the heart may depend on some other, latent, inflammation or as yet unevolved disease; and this excitement, coupled with a modified state of the blood, may suffice to generate a murmur. Thus, occasionally, at the outset of pneumonia, of the exanthemata, and of any inflammatory state, in persons whose blood chances to be in the least spanæmic, murmur occurs. But such murmur, purely of blood-origin, is necessarily systolic and basic; if there be new murmur, either systolic or diastolic at the apex, or diastolic at the base, a complication of acute endocarditis must be admitted; if the murmur be basic and systolic only, it may be hæmic, or it may be endocarditic, and consequently the diagnosis must be deferred. Besides, deposition of fibrin may take place acutely amid the cords, or at the edges of the valves, and induce murmur, without the intervention of inflammation.

So far the murmur has, for argument's sake, been admitted to be new; unfortunately the great difficulty is often to determine that it *is* new. Now two cases present themselves here: (*a*) the murmur exists, when the patient is first seen; or (*b*) it is developed, after observation of him has commenced.

(*a*.) A murmur being already present, the circumstances, *within itself*, favorable to recency of origin, are softness of blowing quality, lowness of pitch, systolic rhythm and aortic constrictive or mitral regurgitant mechanism. The circumstances hostile to recent origin are roughness of quality, high pitch, diastolic rhythm (indeed this is absolutely conclusive, unless there be systolic murmur at the same orifice), and seat at the tricuspid orifice; direct mitral murmur, also, I believe, is never recent. The presence or absence of affections, with which endocarditis is commonly associated, furnishes a guide not to be despised; but without caution the observer may readily be led into error, as in acute rheumatism, by too implicit trust in this very guide. The condition of the pulse cannot be confided in for diagnosis.

(*b*.) An endocardial murmur, *developed under observation*, *at the early period of an acute attack*, is almost a sure index of endocarditis; but even here there are sources of fallacy. In the first place appear those just enumerated, on the hypothesis of the murmur being by admission new, and yet of hæmic origin. In the second place, general collapse and failure of the heart's power may, on the first examination of a patient, prevent a given murmur, of which the physical chronic conditions exist in perfection, from being heard. Reaction takes place, and a murmur becomes audible; that murmur may be chronic mumur solely, or it may be an acute and a chronic murmur combined; but it is *not* that, which it would alone seem to be, namely, an acute murmur *solely*.[1] Again, to have value as a positive sign of recent endocarditis, the murmur must be developed at an *early* period of acute disease; if towards the close, it is almost invariably systolic in time, basic in seat, and a consequence of spanæmia. Further, the possible occurrence of murmurs of dynamic mechanism, muscular and valvular, must not be forgotten. Not only in chorea, but in other nervous diseases, and in nervous conditions supervening in the course of acute maladies in general, is their occurrence a possibility, nay a probability. Such dynamic murmurs are generally systolic and seated at the apex; but there is much reason to suppose that murmurs may also occur dynamically at the base from perverted action of the sigmoid valves.

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[1] Kernis, U. C. H., Females, vol. ii. p. 237. The statements in the text are made on clinical and *post-mortem* evidence.

If during the course of an acute febrile disease, endocardial murmur changes in site and rhythm, this is a very strong, though not an absolute, sign of its dependence on recent inflammation; and, admitting its value, such change is, we have seen, at the least very rare. Again, if a murmur of a certain site and rhythm disappear and return within a short period, the changes might seem proof positive of recency of the cause of the original murmur; but they are not so, at least of murmurs of all varieties of localization. Thus, I have known a direct mitral murmur, essentially organic and attended with great constriction of the orifice, come and go from one day to another: some dynamic conditions must now have obstructed, now have promoted, the occurrence of the murmur.

And there is yet another possible cause of error; an endocardial murmur appears sometimes to occur, according to the testimony of Drs. Latham and Stokes, shortly before death from the impediment of coagulated blood. Fortunately this *præ-mortem* murmur is of rare occurrence: how, if at all, it may be distinguished from an endocarditic one, remains to be ascertained.

So far intra-cardiac murmur has been assumed to be unfailingly present. But may it not be absent in acute endocarditis? The conditions producing murmur are roughness of valves, lymph among the tendinous cords, insufficiency of valves from puckering, and notable roughness, from lymph, of the ventricular surface: now as endocarditis may exist without any one of these anatomical effects, the inference is unavoidable, that it may occur without murmur. I have seen distinct patchy redness with thin films of lymph on the ventricular endocardium, where there had been recent excitement of the heart without newly-developed murmur.[1]

Endocarditis will be distinguishable from pericarditis by the less amount of pain, by uneasiness not being increased by a deep breath, cough or change of posture, by absence of præcordial tenderness, by the less amount of labored breathing, and by the general difference of the physical signs. The distinctions of endocardial and pericardial murmurs, and the alleged value of stethoscopic pressure as an aid in diagnosis, are elsewhere considered [240, 241].

The formation of polypoid concretions may be very strongly suspected if, with the *sudden* appearance of the symptoms enumerated above, a notable increase in the area of percussion-dulness be detected. But rupture of a sigmoid valve, or of a tendinous cord, will produce closely the same effects.

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[1] "Shreddy lymph on surface of left columnæ carneæ recent," without change in character of pre-existing chronic murmurs. Kernis, U. C. H., Females, vol. ii. p. 244, March, 1847.

518. *Prognosis.*—Considerable difference of opinion, not only in regard of minor but of major points, may be traced in the statements of authors concerning the ultimate effects of endocarditis. The results, that have fallen under my own observation, may be set down as follows:—

(1.) Very serious valvular disease, followed by implication of the heart's substance, and all their combined consequences: the ensuing affections of the heart are, in their order of frequency, eccentric hypertrophy, simple hypertrophy, simple dilatation, and, in infinitely rare cases, eccentric dilatation.[1] (2.) Slight valvular disease, with habitual palpitation. (3.) Slight valvular disease, with palpitation under excitement. (4.) Simple murmurs, without any positive cardiac functional disturbance: no morbid palpitation occurring even under severe exercise: this is the most favorable result that I have actually met with. An indubitable endocarditic murmur, holding on from the beginning to the end of the acute disease, never, so far as I have known, totally disappears. The principal sources of possible mistake in this matter deserve enumeration, and have appeared to me to be the following: Temporary obscurity of a recent mitral murmur sometimes arises from the weakness of the heart attending convalescence, and leads to the idea that the murmur is gone, or will go, completely: as the general vigor improves, the murmur recovers its distinctness. Error is sometimes committed too in regard of this point, from change in position of the maximum site of murmurs, especially of the mitral regurgitant variety. And, again, a murmur of anæmia, arising in the advanced period of the disease, is sometimes mistaken for one of really inflammatory mechanism. (5.) On the other hand, death is rare from acute endocarditis alone; still, the disease does occasionally kill, both by secondary impregnation of the blood, and by serious obstruction of the intra-cardiac circulation.

519. *Treatment.*—The ultimate dangers of acute endocarditis are extreme; they involve the ideas of various forms of valvular disease combined with altered dynamism and oftentimes changed statical conditions, both of capacity and of substance, of the heart itself. Acute endocarditis is then a disease in which it is in the highest degree desirable to promptly exercise a controlling power, if we possess this. But, I confess, I have never had satisfactory proof, either from observation or reading, that lavish bloodletting and mercurialization really stay the course, or move the proceeds, of the inflammation-process in this locality; while at the same time no other agencies have even been asserted to possess the

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[1] The relationship between the part of the heart affected and the diseased orifice will be considered with valvular affections generally; for the endocarditic origin of the valve disease gives no *special* character in this way.

powers in question. I believe, consequently, if the direct and instant suspension of acute endocarditis by art be a possible feat, the means of accomplishing it are reserved for future discovery.

Meanwhile it appears to me the course of treatment should be that recommended for acute pericarditis.

If there be reason to suspect the formation of intra-cardiac blood-concretions, the rapid pouring in of liquor potassæ and bicarbonate of potash seems, theoretically at least, worth trial; if there be sinking tendency, sesquicarbonate of ammonia may be given at the same time. Sinapisms should be applied to the extremities. I have seen temporary relief obtained by cupping over the heart: but the patient's strength is rarely, when such obstruction occurs, in a condition to bear the loss of even a few ounces of blood; and the application of Junod's cupping apparatus to the lower extremities is a safer measure.

When the affection seems lapsing into the chronic state, iodide of potassium and liquor potassæ, with bitter tonics, become the best remedies; and iodine-inunctions over the cardiac region appear occasionally useful.

B.—CHRONIC ENDOCARDITIS.

520. Chronic endocarditis is anatomically signified by thickening and opacity of the membrane,[1] uniformly or patchwise—the surface being perfectly smooth or slightly and irregularly puckered. Successive strata, thin, opaque, faintly yellow, and filmy, may be peeled off from the free surface; or minute masses of hard, elevated, yellowish induration-matter separated with the point of the scalpel, leaving the endocardium, apparently uninjured, beneath.

The valves suffer variously. Thickening by infiltrated induration-matter, with hardness, opacity and puckering of their substance, sometimes associated with diminished depth from acute destruction or chronic contraction, are the most common appearances. Calcification, or pseudo-ossification, may ensue in patches, nodules, or spiculæ. Adhesion of the divisions of a valve to each other, or to the neighboring arterial or endocardial surface, is sometimes seen: the former, most common in the aortic valves, may cause them to pouch in the wrong direction; but such adhesion, converting them into a single indurated ring, occurs to perfection in the mitral valves also. Thickening with shortening of the mitral cords, sometimes conjoined with special thickening of the endocardium of the papillary muscles and cirrhosis of these muscles themselves, is clinically one of the most important of chronic changes.

Warty fibrinous products, polypoid or sessile, soft, firm, or calcified, form on the valvular surfaces exposed to the blood-current. The free edge of the mitral, less frequently of the tricuspid valve, frequently presents a semi-transparent beaded thickening, which is not clearly inflammatory, or even morbid.

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[1] **The student must not forget the natural excess of opacity and thickness, particularly as age advances, in the endocardium of the left auricle.**

521. *Symptoms.*—Although, very possibly, certain uneasy sensations about the heart [321] and proneness to palpitation, may depend upon chronic changes in the ventricular or auricular endocardium, yet there is no surety of knowledge on the point. Hence, in point of fact, chronic endocarditis is solely known clinically by its effects on the valves and orifices of the heart; and there is nothing in the statical or dynamic characters of valve-disease originating in inflammation, distinguishing it from that produced by other causes. For the clinical history of chronic endocarditis the reader is consequently referred to the section on valvular affections in general.

III.—Endo-Pericarditis.

522. The combination of the two inflammations, as each separately, may exist in the acute and chronic forms.

ACUTE ENDO-PERICARDITIS.

523. Acute inflammation in a certain proportion of cases attacks both membranes of the heart, either, to all appearance, coetaneously or consecutively.

524. The relative frequency of the double inflammation, and of the two singly, has been variously estimated by different persons. Rheumatic endo-pericarditis is, if Dr. Latham's returns be accepted, somewhat more frequent than pericarditis alone, greatly less frequent than endocarditis. But it is difficult to resist the conviction that the great excess of endocarditic cases in part depended on old intra-cardial murmurs being classed as new; especially as the results of other observers do not tally with those of Dr. Latham.

There are two methods of determining the question of frequency, here started—the clinical and the anatomical. Now both have their difficulties. Thus, in respect of the clinical method, it is certain that friction-sound of pericarditis often renders the detection of existing endocardial murmur difficult or impossible: and that a large amount of pericardial fluid also commonly weakens endocardial murmur, if present; while the pressure of such fluid may, very possibly, in some rare instances, create an obstructive intra-cardiac murmur quite independently of endocardial inflammation [243]. The signs and effects of endocarditis on the other hand cause no difficulty in the discovery of pericarditic signs. In other words, pericarditis obviously existing, endocarditis may very well be present, though its signs are imperfectly discoverable; whereas the converse does not hold good.

So far, then, as these points go, the tendency of clinical observation would be to under-estimate the relative frequency of endocarditis. Besides, as I have already mentioned, acute endocarditis, anatomically demonstrated, does not of necessity produce murmur.

But on the other hand, the similarity of old structural, as well as of certain dynamic and anæmic intra-cardiac murmurs, to those freshly endocarditic, helps, though in a fallacious fashion, to balance the account.

In respect of the anatomical method, endocardial thickenings have sometimes, though purely hypertrophous, passed for inflammatory. *Vice-versâ*, I have strongly suspected in some few instances that endocarditis had really existed, but all congestive state had disappeared before death, and any exudation-products been washed away by the blood-current.

Making such allowance, as I have been able, for these conflicting sources of embarrassment, I find my hospital experience, both in acute rheumatism and in Bright's disease, gives the first place in frequency to pericarditis, the second to endo-pericarditis, and the last, *longo intervallo*, to endocarditis alone.

525. *Prognosis.*—The *immediate* prognosis of the double inflammation is worse, but not very seriously worse, than of pericarditis alone—materially worse than of endocarditis alone; the *ultimate* prognosis of the combined inflammations is very sensibly more serious than of either separately. As we have already seen, it is coexistent valvular mischief that seems really to entail the evils assigned by some writers to pericardial adhesions.

CHRONIC ENDO-PERICARDITIS.

526. The clinical history of this state will, in its practical aspects, be found under the head of Valvular Diseases.

CARDITIS OR MYO-CARDITIS.

527. Inflammation of the muscular structure of the heart, like that of the membranes, is met with in the condition of an acute and of a chronic disease.

ACUTE CARDITIS.

528. Acute carditis presents itself under four conditions, sufficiently different from each other to require separate consideration.

529. *First. Acute carditis, as an attendant on endo-pericarditis.*—The anatomical characters of this form of the disease are well known; they are of tolerably frequent occurrence, on a limited scale, in the strata of fibres nearest the inflamed membranes, especially the pericardium. I have seen exudation-corpuscles and pus-cells amid the fibres under these circumstances.

530. But clinically, the effects of such carditis are not understood; whatever they are, they are lost in the more striking phenomena of the membranous inflammations. Possibly great weakness and fluttering character of the pulse may sometimes be due to inflammatory softening of the left ventricle, in cases of pericarditis without much exudation or fluid effusion, and therefore without an efficient me-

chanical cause of obstructed action. But, although this view of an intrinsic muscular failure has been adopted by Dr. Stokes, it would apparently prove immensely difficult to show satisfactorily that the weakened action does not really arise out of the influence, either direct or reflex, exercised by the inflammation on the nerves of the heart (*vide* Cardiac Paralysis). And a further ground on which I raise this objection is, that in certain indubitable cases of limited purulent infiltration of the heart, the action of the organ has not been found at the outset of the attack either notably deficient in vigor, or seriously irregular in rhythm. I of course refer to cases wherein the absence of acute pericarditis is positively averred by their narrators.

531. *Secondly. Idiopathic and primary general inflammation of the muscular and connective tissues of the heart.*—This is an affection, to say the least, of extreme rarity.

532. *Anatomical characters.*—Vascular injection, deepened color, with crispness, not real firmness, of the texture, seem the earliest alterations. Exudation, sero-sanguineous, or of lymph, or of pus, either in the form of infiltration or abscess, ensues; the texture softens even to pulpiness, and commonly of pale yellowish, grayish, or grayish-brown hue, may, if there be much infiltration of blood, acquire a blackish tint. If there be local softening and abscess, the part affected may bulge outwards, and actually give way into the pericardium. Ulceration extending a certain depth into the wall of a ventricle, or perforating either the pericardium, or the ventricular or auricular septum has rarely, gangrene still more rarely, been seen. The left ventricle is the most frequently affected.

The pericardium seems almost always to undergo secondary implication; the endocardium often escapes, a fact the more strange, as there is strong ground for believing that carditis originates in some special poisoned state of the blood, and is never truly a primary idiopathic disease.

533. The narratives of the few cases of general carditis on record furnish no clue, at least of trustworthy character, to its ætiology. Of the influence of age nothing is known. Hitherto the male sex has furnished the larger share of cases.

534. *Symptoms.*—The clinical history of general carditis is at present singularly imperfect. The rarity of the affection, and its almost constant association with other grave cardiac disease, acute or chronic, or both, serve to explain this imperfection. The only local symptoms appear to be weakness and irregularity of impulse, without positive pain or even uneasiness in the præcordial region, unless where pericarditis coexists. Pyrexia of adynamic character, with a pulse rapidly becoming feeble and irregular, coldness of surface and anxiety of countenance, indicate the gravity of the affection.

535. Of the physical signs little can be said. There is no reason why the percussion-results should deviate from the normal stan-

dard; and in the muscular inflammation itself there exists no efficient cause of intra-cardiac murmur. Impulse excited at first, quickly growing feeble, irregular in rhythm, and unequal in force, is in point of fact the only altered physical condition theoretically to be anticipated.

536. In some cases the heart-disease has been thrown into the shade by the predominance of cerebral and other symptoms, indicating some form of poisoning of the blood, allied to or identical with pyohæmia.

537. *Diagnosis.*—I am not aware that the existence of carditis has ever been diagnosed; nay more, it would appear that the symptoms, the affection has really induced, have, during life, been referred, to the satisfaction of the observer, to some other state which post-mortem examination did or did not prove to be present. I have no personal experience of the disease; and can only suggest the following *à priori* guide to its detection. If grave general symptoms of adynamic pyrexial character coexisted with a state of heart's action, at first excited, and then rapidly losing power, while the absence of endocarditis, pericarditis, typhoid (Peyerian) fever, and other adynamic pyrexiæ, could be made matters of demonstration, a fair suspicion, but nothing more than this, might arise of the existence of carditis.

538. *Thirdly. Partial carditis.*—Partial carditis sometimes occurs, producing abscess, ulceration, and rarely actual gangrene; but of these states no positive clinical signs are known. If perforation or rupture occur, as they sometimes do, the symptoms will vary with its direction. If the septa be perforated, sudden cyanosis may follow, or, it is alleged by Bouillaud, no particularly serious symptoms ensue. The effects of rupture of the heart into the pericardium will be elsewhere considered.

539. *Fourthly. Secondary cardiac abscesses.*—Minute collections of pus form in the heart's substance in a certain proportion of cases of pyohæmia and phlebitis, greatly less frequently, however, than in the lungs and liver. Here, as in other organs, these so called abscesses are often really composed of liquefied exudation-matter alone. The symptoms are yet to be worked out.

CHRONIC CARDITIS.

540. Shortening with thickening of the papillary muscles and columnæ carneæ, and infiltration of their substance with induration-matter, due to a chronic low congestive process, mainly derives interest from its interference with the closure of the mitral, in very rare instances of the tricuspid, valves. There is no character in either a mitral or tricuspid regurgitant murmur, distinctive of this special mechanism. (*Vide* CIRRHOSIS OF THE HEART.)

§ V.—CARDIAC HEMORRHAGES.

I.—Intra-Muscular Hemorrhages.

541. Circumscribed extravasations of blood into the muscular substance occur to very various amounts. The fluid may form mere specks, or apoplectiform nodules as large as a walnut. Or portions of tissue may be infiltrated with blood: the implicated muscular substance is then softer than natural; and, as deficiency of consistence may be traced beyond the limits of the infiltrated part, it seems obvious that a softening-process takes the lead in the morbid changes. This softening involves the minute vessels, hence the extravasation.

I am uncertain whether this form of local softening of the heart with blood-infiltration ever lays the groundwork of true sacculated aneurism; but there is no doubt rupture or perforation of the ventricular wall may ensue. In both cases escape of blood into the pericardium follows; if the opening be at all large, instantaneous death is the necessary result; but if the communication be small and sinuous, the blood may make its way outwards as it were *guttatim*, and forming a layer of coagulum on the surface, temporarily close the external opening.

542. Distinctive symptoms of intra-muscular hemorrhage remain to be discovered, if, indeed, any such really exist. An individual previously known to have a weak and not enlarged heart, and to be free from aneurism, and instantaneously cut off by syncope and presenting *post mortem* the percussion-signs of accumulation in the pericardium, might be suspected to have perished from hemorrhagic rupture of a ventricle; but obviously such mere suspicion would be worth nothing in a medico-legal point of view.

II.—Hæmopericardium.

543. (*a*.) The effusion of inflammation is sometimes so much stained with blood, as to entitle the disease to the name of hemorrhagic pericarditis. It seems probable that such escape of the blood-disks does not take place from the vessels, unless the constitution of the blood itself be affected. I have seen this variety of inflammation well marked in pyohæmia. But no signs or symptoms are known, whereby the hemorrhagic addition to the effusion might be recognized during life. Neither is there any evidence to show that, in the event of recovery, the material of adhesion will be of different character from that observed in the more ordinary class of cases. The quantity of blood is sometimes very considerable, quite enough to give a deep red color to all the fluid in the sac; were paracentesis of the pericardium performed in such a case, the operator would very probably be led, for a moment, to believe that he had punctured some important vessel.

544. (*b*.) Hæmopericardium may be caused by wounds or by

ruptures of the heart itself, by rupture of an aneurism of the aorta or of the heart, by rupture of a coronary artery or vein, or by the giving way of cancerous substance. In all these cases the result is almost instantaneous death, from mechanical obstruction of the heart's action. Some of these cases will again be referred to; the rest are devoid of clinical interest.

545. (*c.*) The pericardium is pretty frequently the seat of extravasation of blood in cases of scurvy—especially in some particular regions of the world. Thus in certain parts of Russia, scorbutic hæmopericardium seems as thoroughly endemic as hæmaturia in the Mauritius.

546. The effusion of blood into the pericardial sac occurs with or without previous scorbutic symptoms, and the attack may be sudden, or so gradual that attention is scarcely drawn to the heart. Præcordial oppression, without pain, or tenderness, and great dyspnœa, seem to constitute the main symptoms; the physical signs will, of course, be those of a pericardium distended with fluid.

547. The more frequent termination is by death: but recovery sometimes takes place by absorption of the blood—the anatomical conditions remaining, being very assimilable to those of chronic pericarditis. Indeed, it seems highly probable that the disease is from the first sub-inflammatory.

548. The treatment, locally, is by cupping—constitutionally, that of the blood-disease present. In the majority of a number of cases, in which paracentesis was performed by Russian physicians, the ultimate result was unfavorable; but in all the immediate relief was extreme—the patient seemed temporarily endowed with new life: and two cases are given of complete recovery. In one of these, related by M. Karawajew, three pints and a half of fluid are stated to have been removed from the pericardium; pneumo-pericardium followed; five months after the operation the patient was considered well.[1]

III.—HÆMO-ENDOCARDIUM.

549. Extravasations of blood under the endocardium in points, maculæ or actual ecchymosis, are by no means excessively rare. They sometimes occur to a notable extent in cases of protracted obstruction to the movement of the blood through the right cavities; and constitute one of the anatomical sequelæ of congestion of the heart's substance [450]. Nor can there be any doubt that these minute hemorrhages play a certain, though not easily definable, part in working out the ultimate effects of muscular congestion.

550. Clinically this matter is a blank: there are neither known symptoms nor signs of this form of hemorrhage.

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[1] British and Foreign Medical Review, July, 1841.

§ VI.—ALTERATIONS OF SECRETION.

I.—Œdema of the Heart.

551. I have seen the heart's substance of watery look, and distinctly infiltrated with serosity, both in cases of chronic pericarditis of the persistent effusion-form [500], and in cases of general dropsy. Nevertheless, ill provided, as it is, with connective tissue, the heart rarely becomes œdematous to any extent. In chronic pericarditis, it is rather the intervening texture between the heart and its inner and outer membranes, than its own substance, that undergoes infiltration.

552. In the present state of knowledge these conditions are merely anatomically interesting. Yet it seems probable that œdema, carried to any amount, may interfere with the heart's contractions.

553. Serous infiltration under the endocardium and between the layers of the sigmoid and of the mitral valves has also fallen under my notice; but I know nothing of either state clinically. It seems quite certain that such a condition of the sigmoid valves must alter the quality and tone of the second sound; whether infiltration can ever reach such a degree as to seriously impede the closure of the valves remains to be determined.

II.—Hydropericardium.

554. Hydropericardium, or dropsy of the pericardium, may be of active, passive, or mechanical origin.

555. (*a.*) Active hydropericardium is very rare: I have, however, in some instances of Bright's disease, known the pericardium fill with fluid—the symptoms indicating an irritative state, while the signs (and hence, I assume, the anatomical conditions) of pericarditis were wanting. I once saw a case which suggested to my mind the question, whether in true hydropericarditis, the plastic material might not be completely absorbed, and the serosity left behind—constituting a sort of sequential active hydropericardium. But I have no positive answer to supply; and possibly, the case referred to was one of active dropsy alone from the first. When hydropericardium is active, it may be the sole dropsy in the body.

556. (*b.*) Passive hydropericardium occurs as a phenomenon of general dropsy—very rarely unless double hydrothorax be already present. The quantity of fluid (colorless, straw-colored, or slightly blood-stained, but without lymph) is generally moderate—from eight to twelve ounces; I have never seen more than the latter quantity.[1]

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[1] A case recorded by Corvisart (Maladies de Cœur, 2ème édit., p. 52), where "about four pints, or eight pounds, of clear greenish serosity," were found in the pericardium, seems to have been one of chronic pericarditis. The distension which the sac must have undergone is the more remarkable, as the membrane is stated to have been thickened.

557. (*c.*) Mechanical hydropericardium has, in some very rare cases, been traced to pressure of aortic aneurism and carcinoma on the great veins, and to morbid states of the coronary veins.

558. The mechanism of hydropericardium is sometimes very difficult of explanation. Thus in a phthisical male, aged twenty-four, cut off in about four hours by perforation of the right pleura and pneumothorax, with little pain, but intense dyspnœa and general distress, the pericardium, pyramidally distended up to the second rib, was found to contain nine ounces and a half of clear faintly yellowish serosity, of specific gravity 1013, neutral reaction, and giving one-third albumen on boiling: there was not the slightest vascularity of, or lymph upon, the serous membrane, which simply looked macerated.[1] The lung was flattened against the side, but sufficient time had not elapsed for the occurrence of pleuritic changes. Could this hydropericardium have been the result of the sudden pressure of, and obstructed circulation in, the right lung with its sequential obstruction in the right heart and coronary veins? It is true, hydropericardium does not appear in the narratives of cases of phthisical perforation; but death rarely takes place so rapidly as this: in truth I have neither met with, nor heard of, a strictly parallel instance. Now, in cases of the ordinary class, where life is preserved for at least thirty-six or forty-eight hours, the first shock of the mechanical difficulty in the course of the cardiac circulation has had time to pass away, absorption has had time to commence, and may before death have removed, or nearly removed, the effused serous fluid.

559. *Symptoms.*—Hydropericardium, however originating, has few subjective symptoms: acute pain and tenderness are altogether, and palpitation commonly wanting; sensations of weight and oppression are alone complained of. The physical signs are in the main those of hydropericarditis. But there is no friction-sound, no præcordial bulging, and the apex of the triangular dulness (mainly because there is less fluid, but partly because there is no inflammatory relaxation and rarefaction of the sero-fibrous membrane) does not mount so high as in many cases of pericarditis. The visible impulse may be very perfectly undulatory.

560. The general symptoms are those of the disease on which the local dropsy depends. The pulse is not necessarily irregular, the quantity of fluid being commonly insufficient to affect the heart's action very seriously; while the constrictive influence of exudation-matter, and any dynamic perversions of muscular action, dependent on contiguous inflammation, are altogether wanting. Orthopnœa may occur; but if so, as far as I have seen, double hydrothorax exists to share in its production.

561. *Treatment.*—Diuretics and hydragogues seem to have less effect on this dropsy than on others: such medicines will, however,

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[1] Imlach, U. C. H., Males, vol. viii. p. 402.

of necessity, be tried, were it only for the removal of the usually concomitant dropsies. Cautious cupping, or dry-cupping over the heart, would be advisable, if the symptoms became urgent. A blister has sometimes appeared to me useful.

Paracentesis has been performed, with temporary relief; but unless the primary disease be removed, of course the fluid will be reproduced. The suggestion of Laennec, that slightly irritant fluids should be injected into the pericardium, so as to excite an inflammation that might, by causing adhesion, prevent the recurrence of the disease, has acquired more claim to serious consideration, since the experiment of injecting an ioduretted solution in hydropericarditis has been successfully tried.

III.—PNEUMOPERICARDIUM.

562. Pneumopericardium is the term devised to signify the presence of gas in the pericardial sac—no other abnormal product or appearance being discoverable. The state may conceivably be of perforative or secretive mechanism.

563. Now pneumopericardium must exist temporarily, be it for ever so few minutes, as the sole result of perforative communication between the pericardial sac and any hollow viscus containing gas; but in this isolated state it has never been observed—pericarditis having supervened before clinical examination has been made.

564. As to secretive or idiopathic pneumopericardium, I am not aware that its existence has ever been conclusively shown after death, much less that it has been diagnosticated during life. Laennec's expressed conviction that in almost all cases, when the heart's action is heard at a distance from the body, the cause of the phenomenon is a temporary development of gas in the pericardium, cannot at the present day be received.

IV.—PNEUMO-HYDROPERICARDIUM.

565. Laennec taught that the effusion of air and serum into the pericardium may occur during the closing struggle in all diseases. He states he had sometimes announced its presence, on the evidence of increased resonance over the lower part of the sternum, and of præcordial fluctuation-sound caused by the heart's action and by deep inspiration.

There is reason to believe Laennec deceived himself as to the frequency with which gas and simple serum are to be found in the pericardium. For my own part I have met with few positive examples of the kind—perhaps I have not been sufficiently watchful. Here is an instance of the kind occurring in a man, aged forty-seven, cut off by phthisis, which had largely excavated the left lung; the following conditions were noted:—

"P. M., forty-six hours after death, June 24, 1858, weather dry and hot.—Pericardial sac considerably distended with gas, giving true tympanitic note on percussion; none of the gas forced away by percussion" [hence the inference that the pericardium was not perforated]; "a small opening being made in front of sac, this gradually collapses, no fluid escaping; upper edge of the sac reaches the level of the first rib; the point of the heart corresponds to the fifth interspace; five and a half ounces of blood-stained serosity in sac, without a particle of lymph; the gas having once escaped, no bubbling through the fluid lying in the back part of the sac follows; the fluid in the sac acid at first; same litmus paper, plunged into it again shortly after removal of the fluid from the sac, becomes blue after a moment's exposure to the air."[1]

Now there were sundry evidences of putrefaction in different parts of the body; still, much more advanced cardiac decomposition is frequently met with unaccompanied with gas in the pericardium. Hence there must have been something special in the case.

V.—PNEUMO-HYDROPERICARDITIS.

566. Gaseous fluid may conceivably accumulate in an inflamed pericardial sac in three different ways. (*a*) Gas may appear as an actual formation by the irritated membrane; (*b*) it may result from decomposition of liquid effusion; or (*c*) be conveyed into the sac by perforation, traumatic or diseased, from a neighboring hollow canal or viscus.

567. (*a*.) Dr. Stokes briefly describes a case of pericarditis, in which for three days metallic guggling and crepitating sounds were heard over the cardiac region in accompaniment with amphoric and cracked-metal note on percussion—the stomach not being distended with air and lungs and pleura unaffected. The heart's sounds were audible at a distance. It seems equally difficult to contest the dependence of these phenomena on the presence of air in the sac, and to explain such pneumatosis by acknowledged pathological laws. There was no *post-mortem* examination.

568. (*b*.) The fluid of pericarditis, fetid and decomposed, has been known to furnish gas during life. In a case of this sort, observed by M. Bricheteau, the heart's action was accompanied with a "sound like that of a water-wheel." The actual presence of gas in the sac was ascertained after death.

569. (*c*.) In the singular case of traumatic communication between the œsophagus and pericardial sac, at some of the particulars of which I have already had occasion to glance, the following were the main physical signs I succeeded in substantiating. The percussion-note over the heart-region was purely tympanitic, not in the least tubular or amphoric—a quality I take as demonstrating that the proportion of gas to fluid in the sac was largely in excess. No guggling noise accompanied the cardiac action, nor were the heart's own sounds, or the existing friction-murmur, particularly loud. The really distinctive phenomenon consisted in the change of position of tympanitic and dull percussion-sound, within the area of the

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[1] Thunder, U. C. H., Males, vol. xv. p. 79; also Crocott, U. C. H., Males, vol. xviii. p. 113.

cardiac region, according as the posture of the patient was changed from one to the other side.[1]

Dr. Graves records a case of fistulous communication between a hepatic abscess and the stomach on the one hand and the pericardial sac on the other. Loud metallic ticking with each stroke of the heart, combined with friction-sounds and a noise like emphysematous crackling, were the signs of the pneumo-pericarditis following perforation. Dr. M'Dowel has observed a case of communication between a cavity in the left lung and the pericardium: metallic tinkling, amphoric buzzing, and splashing of fluid were caused by the action of the heart.

§ VII.—ALTERATIONS OF NUTRITION.

I.—Atrophy.

570. Atrophy may be limited to some portion of the valvular apparatus, or to the muscular structure.

A.—Valvular Atrophy.

571. (*a*.) When the cordæ tendinæ of the mitral valve are shortened and extremely thin, they are, probably, purely atrophous. The larger tongue of this valve is sometimes simply defective in size, without obvious puckering, or other evidence of past inflammation. In both cases, regurgitation may occur. Reticulation of the mitral valve is rare.

(*b*.) The tricuspid valve is sometimes at once deficient in depth, its substance thin and papery, and its cords excessively delicate—conditions apparently proving deficient nourishment, and the first of them certainly tending to promote the occurrence of regurgitation through the orifice.

(*c*.) The sigmoid valves, both pulmonary and aortic, may be thin, and papery; whence a sharp clicking state of the second sound, but no actual disturbance of the heart's action, ensues.

572. These valves are, besides, the tolerably frequent seats of so-called reticulation or cribriform perforation. The minute foramina giving the sieve-like appearance are almost always close to the free edge of each valve, except at the spot corresponding to the corpus Arantii; they vary in size from that of a pin's head to a slit comprising the entire length from the corpus Arantii to the attached edge of the valve; they may be many in number, or but one; and vary in form—round, oval, slit like. Sometimes the perforation is imperfect, the intra-serous tissue only being wanting, while the proper endocardial structure seems sound. All the cardiac textures may be otherwise thoroughly natural, even when this reticulation is carried to an extreme point.

M. Bizot[2] was the first to notice that the pulmonary and aortic valves presented this kind of defect with about equal frequency. He further ascertained that it is more frequent in males than females; and that, rare before the age of fifteen, it suddenly increases in frequency at and just after that age, and ceases to grow more common at and after the age of forty.

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[1] Ramo Samee, U. C. Museum, No. 3859.

[2] Mémoires de la Soc. Méd. d'Observation de Paris, t. i.

573. Now, I speak of reticulation of the valves under the head of atrophy, to avoid multiplying divisions; it seems to me very doubtful whether it be truly, or at least simply, atrophous. I can scarcely conceive it to be a fatty form of local waste, because it implicates the two sides of the heart with such closely equal frequency. On the other hand I confess I cannot accept as satisfactory, ingenious though it be, M. Bizot's theory, ascribing the condition to rupture from extension by the rapid increase in the heart's dimensions, which sets in at the period of life when reticulation first becomes common.

574. *Symptoms.*—Interesting enough in an anatomical point of view, this sieve-like condition proves clinically unimportant. It produces no subjective annoyances of any kind. On first thought it seems conceivable this state might cause regurgitant arterial, that is, basic diastolic, murmur. But I have never known a murmur actually so produced; and if its occurrence were usual, the murmur signifying pulmonary regurgitation ought, instead of being one of the *mirabilia* of clinical practice, to be common—seeing that reticulation is very closely as frequent in the pulmonary, as in the aortic, valves. Besides, there is an anatomical cause why, unless in extreme cases, reticulation should have no disturbing influence on the circulation; it affects those parts of the valves, close to their free lunated edges, that lie vertically surface to surface in the centre of the vessel, at the moment of its systole;—these particular portions of the valves have nothing directly to do, physiologically, with the prevention of regurgitation. It is, perhaps, barely possible, however, as already suggested [217], that the peculiar basic sound I have endeavored to represent by the syllable *phwē* .. *tt*, might be thus engendered, reflux of blood taking place at the commencement of the act of closure.

B.—MUSCULAR ATROPHY.

575. (*a.*) The heart is said to be the subject of *concentric atrophy*, when the size and weight of the organ and the capacity of its cavities are alike diminished.

576. I have been led in but a single instance to imagine I had met with an idiopathic example of this affection during life. Here no diathetic disease of any kind existed; the symptoms and signs solely indicated smallness and feebleness on the part of the heart: but I want the *post-mortem* proof of the correctness of the diagnosis. Congenital smallness of the coronary arteries may conceivably act as a cause of such defective nutrition.

577. I have seen an atrophous state of the left ventricle (as others

had done before me), produced by the close embrace of pericardial induration matter. But so far from such embrace habitually entailing atrophy, it seems rather to stimulate the nutrition-process in the organ. Accidental pressure on the coronary arteries probably explains atrophy when it occurs. Atrophy, it is alleged, has sometimes been artificially produced by the means adapted for the treatment of hypertrophy—a statement which, although emanating from Laennec, still requires corroboration.

578. In phthisis and in carcinoma, as originally shown by MM. Louis and Bizot, the weight and bulk of the heart fall, as a rule, sensibly below the average. So true is this, that the sustainment of the heart's weight at the par of health, in persons slowly cut off by either of these diseases, may in my apprehension be looked upon as the equivalent of hypertrophy in people free from the one or the other.

579. No subjective symptoms can be traced with positiveness to this condition of the heart. Palpitation has sometimes been observed, but in carcinoma is probably rather the result of attendant spanæmia than of the atrophy. It would appear that cardiac paræsthesiæ of different kinds may occur;—I have observed the coincidence, but cannot feel sure of the real connection of cause and effect. The pulse is small and wanting in vigor: other characters, appertaining to those diseases, affect it in cancer and phthisis.

580. The heart's impulse is deficient in force, and materially limited in area both to the eye and hand. The extent of præcordial percussion-dulness, both superficial and deep-seated [95], ranges very materially below par. Thus in the apparently idiopathic case above alluded to, percussion of medium force failed to elicit cardiac dulness over a space larger than from one-half to two-thirds of that fairly to be looked for in an individual of the same stature possessed of a heart of normal dimensions. It is important to add, there was neither emphysema, nor any other condition capable of interfering with the heart's percussion, present. Of the character of the cardiac sounds I know nothing of importance. In this case they were not appreciably abnormal.

581. (*b.*) *Eccentric Atrophy.*—When the walls of the heart are greatly attenuated with or without subsidiary dilatation, loss of mass has occurred, and the state may fairly be termed one of eccentric atrophy: the fibre is deficient in firmness. It is singularly rare; and is rarer still in the left than the right ventricle. In the latter situation it probably intensifies some of the effects of the dilatation and tricuspid insufficiency, with which it is commonly associated; but, in point of fact, I have seldom seen atrophy of the kind.

582. (*c.*) *Partial or Local Atrophy.*—This form of mal-nutrition is far from uncommon. In this point of view it would call for full description; but on the other hand, in its anatomical conditions and functional effects, it is so closely associated with certain other

changes of texture, that its history may be most advantageously given with the account of these changes—namely, fatty infiltration, fatty metamorphosis, and cirrhosis.

583. The heart's fibre seems but little susceptible of that intrinsic form of decay, unattended with adventitious production of any kind, known in the voluntary muscles as Cruveilhier's progressive muscular atrophy. Still local waste of analogous character does sometimes appear to occur; but, singularly enough, not when the external muscles are affected. There is nothing known with precision concerning this state clinically.

II.—HYPERTROPHY.

A.—VALVULAR HYPERTROPHY.

584. Thickening, more or less notable, with opacity of both the mitral and aortic valves, is sometimes observed in connection with hypertrophy of the left ventricle, when no anatomical or clinical indications exist of bygone inflammation; and where, in all probability, the thickening results from excess of nutrition consequent on the extra-work entailed on the valves by the muscular hypertrophy.

585. This state of the valves impresses a dull, heavy, clanging character on the valvular portion of the first, and more especially on the second, sound: this is matter of observation. I am not aware of ever having met with a case in which this hypertrophous state was carried to such a point as, unassisted, either to throw a difficulty in the way of the onward current sufficiently serious to generate obstructive murmur, or to interfere so effectually with the closure of the valves, as to cause regurgitant murmur. But it is well conceivable that where such murmurs are produced by other causes, valvular thickening must intensify them.

B.—CARDIAC HYPERTROPHY.

586. In a certain share of cases of hyper-nutrition of the muscular substance of the heart, the affected walls and their inclosed cavities retain the relative proportions of nature—as the former thicken, the latter *pari passu* widen: in such cases the hypertrophy is termed *simple*. Or while the muscular substance of, say, a ventricle increases in mass and density, the area of its cavity disproportionately enlarges: the hypertrophy is then styled *eccentric* or *dilated*. Or, lastly, while the muscular mass grows, it may encroach upon and actually lessen the capacity of the cavity it bounds: hypertrophy of this sort is known as *concentric* or *contracted*.

Hypertrophy may be *general*, that is, affect all the compartments of the heart; in this case it is always, as far as I have seen, of the dilated or eccentric species; or the extra-nutrition may be *limited* to one or two compartments; under these circumstances the species varies.

Whether alone, or in association with other portions of the organ, the left ventricle is by far the most frequent seat of hypertrophy; next comes the left auricle, closely followed in point of frequent implication by the right ventricle; while *longo intervallo* the right auricle completes the series.

The thickness of the wall of the left ventricle may increase from the normal amount of half-an-inch to two inches, according to some observers; I have not known it exceed one and a quarter inch exclusive of the columnæ carneæ. As a general rule, the basic part of the ventricle thickens most, the apex-region relatively least; in some rare cases of old standing aortic regurgitation, however, the precise converse is observed.

The columnæ carneæ, and especially the papillary muscles, thicken variously; if there be notable dilatation they are flattened, and may appear attenuated, though really extra-nourished.

The septum, enlarged similarly, encroaches on the cavity of the right ventricle, which becomes elongated, narrow, and concave, on the side next the septum.

But not only may hypertrophy of a given compartment be or be not associated with contraction or dilatation of its own cavity, with one or other of the three species of hypertrophy in certain other divisions of the organ, but also with simple or attenuated dilatation of certain of those other divisions. The variety of conceivable combinations of hypertrophy and attenuation, of dilatation and contraction, is hence obviously very numerous. Nature, however, actually produces only a limited few of the possible total number. The late Dr. Hope suggested an arrangement in order of frequency of those clinically known, to which I cannot help thinking exception may on more than one point be taken.

As far as my own observation goes, the most common original seat of hypertrophy is the left ventricle; a similar state, carried to much less amount, more or less rapidly, follows in the auricle, while dilatation advances in both. And clinically these morbid conditions may long remain confined to the left side; though before death, generally speaking, the right divisions have become implicated, so as to account for the fact, that general dilated hypertrophy holds the first place in frequency in Dr. Hope's table founded on *post-mortem* examination.

The physical conditions of an over-nourished heart vary of course with the capacity of its cavities. The superficial bulk of the organ does not exceed that of health in the concentric form, equals and exceeds it in the simple variety, more or less enormously outstrips it in the dilated species. In the latter it is that the general mass may be augmented to double, treble, even quadruple the normal amount; I have known the organ weigh forty ounces;[1] and entertain no doubt on the evidence of physical signs, of having met with still greater increase of weight in some instances, where the opportunities for *post-mortem* examination did not present themselves.

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[1] Allen, U. C. H., Males, vol. xi. p. 346.

In simple hypertrophy the special form of the heart is retained; while just in proportion as dilatation is superadded, does the shape become unnatural, spherical, square, or even broader than long. The apex under the latter circumstances is so rounded off that it ceases to be distinguishable.

In regard of position in the chest though the hypertrophous heart is habitually lowered, as an effect of mere weight, it occasionally becomes fixed at the normal level by pleuro-pericardial adhesions. The enlargement then affects the upper limits of the cardiac region; the organ reaches the second cartilage above, and the eighth rib below, stretching vertico-diagonally from nearly two inches to the right of the sternum to two and three-quarter inches outside the vertical line of the left nipple. In very rare instances of dilated hypertrophy, especially where the chest is deficient in transverse width, the heart may literally occupy the greater part of the left lateral regions of the chest and *quoad* percussion-results simulate pleural effusion.[1]

The coronary arteries, one or both according to the seat of increased growth, enlarge. Dr. A. Lee states that the nerves and ganglia in the substance of the heart also increase in mass. Cloetta,[2] following in the inquiry, admits the enlarged size of the nerves, but professes himself in doubt whether the enlargement is due to the actual nervous substance or the fibrous tissue.

587. The walls of the aorta thicken, and the calibre of the vessel widens. The latter change is well shown at the orifice, which, if the right side of the heart be relatively ill-nourished, may very considerably exceed the pulmonary in width. These extra dimensions may certainly be found in the innominate and large vessels at the base of the neck also—and in some instances, which for my part I believe to be rare, the inferior thyroid arteries, according to the testimony of Drs. Parry, Graves, and Stokes, enlarge. It has even been affirmed that the entire arterial system widens; but hypertrophy is commonly a disease of middle and advanced life—in other words of a period when, as to all seeming demonstrated by M. Bizot, the arteries normally increase in calibre. There is no sufficient evidence to show that hypertrophy causes atheroma, calcification, or fissuring of the arteries; but the association occasionally observed of calcification of the pulmonary artery with dilated hypertrophy of the right ventricle (each of them so rare) seems to exhibit a relationship of causality.

588. The microscopical characters of the individual muscular fibre in hypertrophy do not deviate from those of health; and, as the primitive fasciculi do not increase in thickness, new ones are

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[1] C. R., August, 1855.

[2] Virchow's Archiv., 1853.

obviously formed. Production of non-striated fibre, or even of fibre more imperfectly striated than in health, does not occur. The direction of the fibres sometimes undergoes slight change, evidently from lateral pressure of new fasciculi irregularly produced; the change is insufficient to modify on any efficient scale the natural line of action of the fibres [63]. Firm hypertrophy in one part of the walls of a ventricle may be associated with softness and anæmic tint, or with fatty change, in another: an anatomical fact which frequently proves of clinical importance, by modifying the manner of impulse of the enlarged organ. Even in the firm variety, the hardness and tenacity of each individual fibre do not appear to undergo increase: close packing leads to simulation of augmented hardness.

589. *Causes.*—The causes of hypertrophy of the different compartments of the heart may be grouped in the following manner. In some instances the reality of the assumed influence is more or less doubtful—a fact signified in each instance by a note of interrogation.

I. *Causes originating in the system at large.*—Excessive nourishment, especially nitrogenized, combined with free use of stimulants, and sedentary habits; excessive exercise, as pedestrian, rowing (and that of trades straining the upper extremities in excess?).

II. *Causes originating in the blood.*—Uræmia? rheumatic hyperinosis?

III. *Causes originating in the heart itself.*—(*a.*) *Functional:* Excitement of the heart, as the habitual palpitation of prolonged anæmia; irritation of chronic pericarditis. (*b.*) *Mechanical Obstruction:* (1.) *Affecting left ventricle:* Aortic constriction, aortic regurgitation, mitral regurgitation, mitral constriction?[1] mitral regurgitation and constriction combined. (2.) *Affecting right ventricle:* Tricuspid regurgitation, pulmonary constriction, mitral regurgitation indirectly through engorgement of the lungs. (3.) *Affecting left auricle:* Mitral regurgitation and constriction, aortic regurgitation and constriction in much less degree. (4.) *Affecting right auricle:* Tricuspid regurgitation.

IV. *Causes originating in the great vessels.*—Pressure obstructing their interior; smallness or constriction of aorta; aneurism of aortic arch near the heart?? (*vide* Aneurism of Aorta); diminished elasticity of coats of aorta or pulmonary artery, affecting severally left or right ventricle?

V. *Causes originating in the lung-circulation.*—*Affecting right ventricle:* Chronic bronchitis; emphysema; contraction after pleurisy; dilatation of bronchi and cirrhosis of the lung; diminution of cavity of chest, by deformity or by pressure of abdominal tumors? It is possible, too, that the left ventricle may eventually become hypertrophous from the strain thrown on it by the systemic, sequential to the pulmonary, obstruction.

VI. *Causes originating in the kidney.*—Persistent obstruction in its capillary circulation?? [596].

VII. *Causes originating in systemic capillary obstruction.*—Prolonged obstruction of any given mechanism in some large portion of the systemic capillaries? (*vide* V.).

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[1] Mitral constriction may be hypothetically supposed to cause left ventricular hypertrophy, either through the effort of the ventricle to overcome the systolic capillary obstruction consequent on the pulmonary obstruction, immediately induced by the mitral constriction; or through the extra effort made by the ventricle to propel the small quantity of blood supplied by the auricle, to make up, as it were, by force of propulsion, for smallness of supply.

The most easily intelligible of all these modes of production of the disease seems to be that depending on mechanical difficulty at some one of the orifices; but no direct ratio constantly holds between the amount of hypertrophy and of valvular obstruction—showing that even in these cases there is a something beyond mechanical difficulty which contributes its quota of causation.

Males are in a very considerable ratio, at least that of 2 : 1, more subject to the disease than females—probably rather because they are more exposed to its exciting causes, than from any inherent sexual peculiarity. Very much on the same principle is to be explained the increased frequency of the disease with advancing years: well-marked hypertrophy may, however, exist in earliest childhood.

590. *Symptomatology.*—The symptoms, signs, and effects of hypertrophy of the different compartments of the heart differ so materially, that, to avoid confusion, it will be advisable to consider the disease in each situation separately. Nor must it be forgotten that pure and simple examples of hypertrophy are rare; that valvular affections commonly exist to modify both the subjective, and especially the objective, effects of enlargement.

591. I. *Hypertrophy of the general substance of the Left Ventricle*—either pure, or combined with dilatation of the cavity insufficient in amount to throw its own special characters into the shade. In the following description a highly-marked case is taken as the model.

592. *Physical Signs.*—Inspection discloses arching of the præcordial region (especially in long-standing cases, and in early youth), with widening, but without bulging, of the left interspaces, from the third to the seventh. The impulse, increased in extent, especially to the left of the sternum, presents its maximum amount below and about the left nipple, and between this and the sternum; in character it is slow, heaving, and suggestive of pressure forwards steadily against an obstacle; in rhythm regular, unless there be some added morbid state; in force unequal. The amount of force may be sufficient to shake the head of the observer, the trunk of the patient, or the bed even on which he lies; such extreme power of action is rare, unless dilatation be combined with great hypertrophy. Of double systolic and diastolic impulse, I have already spoken [77]. The point of the apex-beat, carried downwards and outwards, may reach the lower edge of the seventh rib (rarely, however, without dilatation), at some distance outside a line let fall perpendicularly from the nipple. In eccentric hypertrophy the extent of visible impulse is much greater; the apex-point may be carried to the seventh space or eighth rib; the impulse may, without much difficulty, be felt in the back; its character is less heaving than in the pure disease, sharper, more knocking, or slapping, and the surface, over which it is perceptible to the hand, proportionally more extensive.

The superficial and deep-seated dulnesses of the heart are both augmented in area, and, probably, in amount also—the parietal resistance is sometimes very notably increased. In dilated hypertrophy, the dulness may reach from the second to the eighth rib—proving marked enlargement in an upward as well as downward direction. Transversely cardiac dulness may stretch from an inch and a half or two inches to the right of the sternum to three inches and even upwards outside the vertical line of the nipple: and want of resonance may be detected in the back to the left of the spine. Nay, more, in those exceptional instances of huge bulk a moment since referred to, the percussion-sound may be toneless and high-pitched all over the left lateral regions of the thorax, and vocal fremitus null, just as if pleural effusion existed.

The rudely-triangular form, natural to the heart's *superficial* dulness (*vide* Diagram, p. 3), gives place to a dulness of somewhat square outline; and the deep-seated dulness is also more right-angled than in health—this latter character, however, is not often to be satisfactorily ascertained.

The state of the cardiac sounds differs with the species of the hypertrophy. First, in the *simple* form the first sound is dull, muffled, prolonged, weakened in some cases almost to actual extinction, directly over the ventricle, the sensation reaching the observer's ear being rather one of impulsive motion than of sound;—under these circumstances a tolerably full systolic sound may, nevertheless, frequently be found at the base and at the ensiform cartilage; the extent of its transmission towards the apex is very limited. The second sound, lower pitched than natural, is fairly loud and of clanging quality. The post-systolic silence is shortened. During palpitation the first sound sometimes becomes comparatively full-toned. Secondly, in hypertrophy with dilatation, the sounds gain greatly in loudness, and extent of transmission, especially if the valves be perfectly healthy and free even from hypertrophous thickening; and the tone of the first at the left apex is notably higher pitched than natural. Reduplication of either sound, sometimes occurring, possesses no special character: it is not common—especially when the inequality of strength in the two ventricles is considered.

Systolic blowing murmur, basic and audible at the second right cartilage, is sometimes heard in cases of pure hypertrophy; nor can it be positively ascribed in all instances to coexistent spanæmia. I have known such a murmur disappear, when the heart had become comparatively quiescent by treatment of a kind depressing rather than otherwise, and wholly non-ferruginous. Hence excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur [204]. Again, hypertrophy may possibly, during the excitement of palpitation, induce mitral regurgitant murmur by disturbing the action of the papillary muscles—especially as these muscles are often relatively nourished more sometimes less, than the rest of the ventricle, sometimes even more or less extensively cirrhosed. Such murmur actually does, as a clinical fact, exist at one time and disappear at others.[1] And systolic basic murmur may also, very probably, be generated in cases of dilated hypertrophy, in consequence of the altered relationship of the aortic orifice to the cavity of the ventricle—altered both in point of size and of direction of the blood-current [188]. In dilated hypertrophy, knocking and rubbing additions to the first sound at the apex, either left or right, are not very uncommon. Possibly, too, the second sound may be, in seeming at least, intensified at the left apex by the abrupt recedence of the enlarged heart from the side during its diastole [149].

The main axis of the heart, when any notable dilatation is conjoined with the hypertrophy, becomes more horizontal than natural. It has been supposed the great vessels may consequently undergo more or less marked twisting at the base, and systolic murmur arise in consequence. I have never known murmur demonstrably thus caused; the effects of lateral detrusion of the heart in pleuritic effusion, while they show such an effect is not impossible, would lead us to expect that it must be very rare.[2]

The respiration at the centre of the cardiac region, that is, the upper sterno-costal angle of the fourth left interspace, is feeble and distant—but not so feeble nor so distant as it would be with an equal amount of percussion-dulness from fluid in the pericardium.

In estimating the dulness really depending on a hypertrophous heart, the observer must bear in mind, that its apparent extent may be, on the one hand, increased by blood-engorgement of the right cavities, by aneurism of the aorta, by indurations in the lungs, pleura, or mediastina, by tumors of the œsophagus even, and by enlargements of the liver, which, by pushing the organ upwards and to the left, widen the area of dulness in those directions;—and, on the other hand, decreased by emphysema and bronchitic distension of the lung.

593. *Symptoms and state of the functions generally.*—The state of the functions may be described as follows, in cases of pure hypertrophy, or hypertrophy without any notable amount of dilatation —the excess of nutrition being either wholly limited to the left compartments, or involving the right to a comparatively slight extent.

(*a*.) The strength does not seriously suffer, unless the disease be carried to a great height; the power of walking and of ascending hilly ground is diminished, not from feebleness, but from the dyspnœa and oppression induced by the attempt. Patients generally lie with the head high.

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[1] Bousey, U. C. H., Males, October, 1850. Slight systolic murmur existed at the left apex in this case when the heart was much excited, disappearing wholly when its ordinary beat had been calmed down by treatment.

[2] Diseases of the Lungs, 3d Am. edit., p. 212.

(*b.*) The color of the integuments varies; if the hypertrophy be pure, the face is florid, if coupled with moderate dilatation, there may be slight purpleness and lividity; but marked purple discoloration does not occur, unless there be very considerable dilatation, valvular obstruction, or pulmonary disease. Hypertrophy of the left ventricle does not *per se* produce œdema of the ankles, much less general anasarca: even hypertrophy and dilatation, unless the latter be in great excess, fails to induce this evidence of systemic vascular obstruction.

(*c.*) The muscles are well-nourished and of good color.

(*d.*) There is not any form of dyspeptic derangement particularly assignable to hypertrophy; the disease may exist for years without materially affecting the digestive powers, provided moderate exercise be taken. Constipation acts as a source of habitual annoyance.

(*e.*) There is more or less dyspnœa, either constant or occasional —in the latter case induced by the most trifling effort: the pulse and respiration-ratio may be perverted in consequence; paroxysms of dyspnœa have not occurred under my observation, unless there were much dilatation, valvular obstruction, or pulmonary disease. Dry cough annoys some patients; I have not observed œdema of the lungs, nor hæmoptysis.

(*f.*) The radial pulse, in no wise peculiar in regard of frequency, and perfectly regular in rhythm, is full, strong, firm, tense, resisting, and prolonged, without jerk or thrill, in the pure disease; if dilatation be superadded, it retains its fulness, but loses in some measure strength and resisting power. It is said not to be increased in frequency, as in health, by change from recumbency to the sitting and standing postures. The action of the carotids is visible; and in aged persons the pulsations of the smaller superficial arteries may be generally distinguished by the eye [251]. Præcordial pain, rare in simple hypertrophy, is not uncommon in the dilated variety, ranging in severity from a slight aching sensation to the severe suffering of pseudo-angina. Paroxysms of such pain may be accompanied with, and probably depend sometimes on, congestion of the lungs and loading of the right cavities of the heart with blood; in some instances they are distinctly traceable to intercostal neuralgia. They rarely attain any notable amount of severity, unless valvular disease coexist.[1] The common action of an hypertrophous left ventricle would be palpitation, if not in frequency, in force, to a healthy person; under excitement, or often without apparent cause, a violent fit of throbbing action comes on—regular, however, or almost so, in rhythm, producing forcible pulsation in the neck and head, with tinnitus aurium.

(*g.*) No change, that I am aware of, occurs in the lymphatic vessels or glands.

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[1] Allingham, U. C. H., Females, vol. xvii. p. 53. But even with this associated condition any near approach to genuine angina is very rare in cases of hypertrophy [433].

(*h.*) I have not known hypertrophy produce albuminous impregnation of the urine, nor indeed any distinctive condition of the fluid—not even notable modification in the quantity, as compared with the amount of drink. The dynamic agencies, which may by possibility affect the renal circulation in cases of pure or dilated hypertrophy, are doubtless on the whole conflicting enough; yet it seems to me that if the doctrines taught of late years concerning the influence of eccentric pressure of the blood in the capillaries of the Malpighian tufts on the amount of water excreted, were perfectly sound, the mean daily discharge of urine ought to range more or less steadily and markedly above par.

(*i.*) It has been said the sexual propensity undergoes increase—a statement I can neither affirm nor deny—but one which seems to have been put forward on theoretical grounds.

(*k.*) Cephalalgia, dull, aching, or throbbing, is of more frequent occurrence than in healthy persons, but by no means a constant symptom; sensations of rushing of blood to the head are common, especially on stooping, and, indeed, on sudden movement of any kind. The intellect is habitually unaffected, as regards any symptomatic state clinically significant: no proof exists of its being brightened; nor, on the other hand, unless towards the close of life, have I found pure hypertrophy render individuals incapable of ordinary mental exertion. A disposition to drowsiness, especially after meals, occasionally, but not by any means with the frequency that might be anticipated, proves a troublesome symptom.

(*l.*) Reflex muscular phenomena, at the moment the patient drops off to sleep, sudden starting of the legs, for example, are not very uncommon; they may possibly be traceable to active congestion of the spinal cord.

(*m.*) The eyes of some patients are bright, full, prominent, prone to injection; and by such persons visual illusions, luminous vision, and muscæ volitantes are frequently complained of. I am not aware that cataract or other textural change in the eye has been found of unwonted frequency in pure hypertrophy. Epistaxis seems to be more usual than in individuals of equal age free from hypertrophy.

(*n.*) Whether sufficient evidence exists of the alleged influence of hypertrophous heart in causing enlargement of the thyroid gland, may perhaps be reasonably doubted. However the statements of Drs. Parry, Graves, and Stokes, concerning enlargement of the gland and of the inferior thyroid arteries as a dependence on permanent excitement of the heart's action, most certainly deserve attention. Dr. Stokes teaches that, though the cardiac disturbance, causing such excitement, be originally functional, dilated hypertrophy is eventually found—that, more common in the hysterical female, thyroid enlargement still does occur in the male—that the enlarged gland pulsates and furnishes the signs of aneurismal varix, all of which signs may disappear with increasing solidity of its structure.

594. *Secondary morbid changes.*—Certain textural changes have been held to follow as a necessary consequence of the influence, long sustained, of a left ventricle over-nourished, and hence, by inference, powerful to excess in its contractions.

595. Hypertrophy of the left ventricle has thus been said to entail increase in weight and substance of the organs generally. Plausible enough though the proposition be, and supported though it be to a certain extent by some statistical returns of Dr. Clendinning, it cannot be looked upon as an established truth. I have seen rare cases in which the weight of several of the organs actually fell below par. But pure hypertrophy of the heart is difficult to meet with; and if fatty atrophy to any serious measure deteriorate the fibre of the generally thickened ventricle, the conditions of the problem are plainly altered.

596. The great frequency with which morbid changes in the heart and the anatomical conditions of the diathetic affection known as Bright's disease are associated, is matter of universal recognition. But opinions are far from being unanimous concerning the relationship of the cardiac and renal states in point of sequence, yet more in point of causation.

Setting aside for the moment those much more common cases where some form of valvular disease coexists with the muscular extra-growth, and confining myself to instances of pure hypertrophy, dilated or not, but unattended by disease of the orifices sufficient to disturb the circulation notably, I find some few instances in my hospital-books where from the clinical course of events, as well as from the *post-mortem* appearances, the renal changes must have preceded the cardiac in order of development —none where the cardiac distinctly led the way—some where the sequence could not be determined. I take it, then, we may admit that the renal disease is not caused by the cardiac.

Is the cardiac *per contra* caused by the renal? Dr. Bright, who first noticed the relationship of coexistence, seems to have supposed the struggle against the capillary obstruction in the kidneys sufficient to account for hypertrophy and dilatation of the left heart. I cannot accept this mechanical doctrine. My cases show me no steady proportion holds between the amount of capillary destruction in the kidney and the cardiac hypertrophy.

Believing, as I have long done, on what appear to me logical grounds, that Bright's disease is primarily a diathetic blood-affection,[1] entailing, like all affections of the diathetic group, secondary alterations in the blood also, I look on the cardiac hypertrophy, occurring in its course, as partly an additional local expression of the main diathesis, and partly as a result of the specific irritation of the blood, primarily and secondarily altered in composition, on the endocardial surface.

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[1] "Bright's disease essentially and primarily a Blood-disease."—Clin. Lect., Lancet, July, 1849.

There may besides be some connection, through the fatty diathesis, between pseudo-hypertrophy or fatty enlargement of the heart and fatty alteration of the kidney; but I have seen nothing demonstrative of any such connection.

597. Hypertrophy of the heart, and especially of the left ventricle, has long been supposed to play a very important part in producing certain acute and chronic textural changes in the brain. The following passage, now some years old, still seems to me to place the actually attained knowledge of the matter in the fairest point of view. In order to avoid the necessity of recurrence to the subject some other conditions of the heart are at the same time considered:—

"Nothing can appear more plausible than to suppose undue force of propulsion of the blood from the heart shall act as a cause of apoplexy, whether this be simply congestive (ictus sanguinis), hemorrhagic, or dependent on red softening. Now, over-forcible propulsion of the blood through the systemic arteries is mainly the result of too energetic contraction of the left ventricles. Hence hypertrophy of the left ventricle has been, by a process of *à priori* reasoning, set down as a cause of apoplexy in general by some persons; by others, of cerebral hemorrhage, in particular; by yet others, of red softening. Again, the doctrine referring white or colorless softening of the brain to deficient nutrition, occurring independently of inflammation, has had many followers. Persons taking this view of its nature are prepared to recognize, in any agency interfering with the distribution of a just amount of blood to the brain, an efficient cause of the disease. Now, constrictive disease of the aortic orifice, and regurgitant disease of the mitral orifice, must have this effect on the cerebral circulation, and so, it is inferred, must act as causes of cerebral softening.

"It has been felt, I may almost say, on all sides, that these views require to be tested by facts; and the facts adduced, as far as they have reached me, may be summarily set forth as follows. First (*a*), in regard of 'apoplexy,' and 'cerebral hemorrhage,' we find writers giving the subjoined numerical results of their observations concerning connection between those cerebral affections and heart diseases.

	Deaths from Cerebral Hemorrhage.	Heart Hypertrophous.
Rochoux	42	3
Andral	17	9
D. Fardel	28	8
	Apoplexy with Hemiplegia.	Heart Disease.
Clendinning	28	15
Hope	39	27
Burrows	34	23
	188	85

"Calculating from the sums of these somewhat contradictory results, there seems presumptive evidence that the heart will be diseased in about 45.2 per 100 cases of apoplectic seizure with sudden hemiplegia. But the precise ages of the persons supplying these figures is unknown. That it was advanced in the great majority of cases may be admitted from the known laws of cerebral hemorrhage; that it was advanced in M. Fardel's patients is certain, for they were all observed at the Salpêtrière, where the rather patriarchal age of sixty years forms a condition of admission; the mean age in M. Andral's cases I calculate to have been 56.2 years. Now what proof have we that the number of hypertrophous hearts in the victims of cerebral hemorrhage, just counted and percented, is greater than it would prove in a like number of aged people, cut off by all diseases, indiscriminately, affecting other organs than the brain? Absolutely none. So far as evidence goes, this seems, on the contrary, to show that the inference of close nexus of cause and effect, in cases where the cardiac and cerebral diseases were found, is illogical. Thus M. Fardel carefully examined the bodies of sixty aged persons (at the same institution, the Salpêtrière), cut off by other than cerebral causes, and found the heart sound in forty-five, hypertrophous in fifteen, of the number. In other words, we may expect that 25.0 per cent. of a mass of aged persons cut off by all diseases indiscriminately (except those of the brain), will have a hypertrophous heart. Now, this proportion of 25.0 per cent. is only twenty per cent. less than that of diseased hearts, furnished by the above series of apoplectic persons.

"I have separated the results of the six authors quoted into two sub-series. The separation is an important one enough. The first sub-series refers especially to 'cerebral hemorrhage' in connection with 'hypertrophy;' in the second, the association is between 'apoplexy' and 'heart-disease,' no precise affirmation being made, or intended, as to the condition productive of apoplexy, or as to the nature of the heart-affection. It is a striking fact that the first sub-series gives a proportion of hypertrophous hearts of only twenty-three per cent., absolutely ten per cent. less than in aged persons wholly free from cerebral affection.

"Let us next examine the question in the converse point of view, and see in what proportion of persons having hypertrophy, or other disease of the heart, as their main affection, cerebral apoplexy supervenes. Here the following numbers are available for our purposes:—

	Disease of Heart.	Cerebral Apoplexy.
Ravier	10	1
Louis[1]	45	0
Blakiston	155	14
	210	15

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[1] I heard this numerical statement made by its author in 1837.

"So that 7½ per cent. only of persons laboring under diseased heart, for a greater or less number of years, become the victims of cerebral apoplexy.

"Secondly (*b*), in regard of cerebral softening, the following figures may be adduced:—

	Brain softened.	Heart hypertrophied, or hypertrophied and dilated.	Aortic constriction.	Mitral regurgitation.
Andral . . .	33	9	3[1]	—
D. Fardel . . .	41	8	—	2
Rostan . . .	18	12	—	—

"From these figures it would follow that 35.9 per cent. of persons having a softened brain had hypertrophy, or hypertrophy and dilatation of the heart, scarcely more than M. Fardel's average for aged persons dying of all diseases indiscriminately. In the series just given, Rostan's figures all refer to old persons, and those of Fardel to persons aged upwards of fifty. The latter observer affirms that he always found the heart healthy in patients cut off by softening before the age of fifty. The mean ages in Andral's cases of softening I have calculated as follow: Where the heart was sound, 49.9 years; where there was hypertrophy, or hypertrophy and dilatation combined, 47.7 years; and where there was constrictive disease of the aortic orifice, 63.7.

"Eight cases have been adduced by Dr. Law, to show that colorless softening of the brain (which he, with many other persons, holds to be allied in nature to gangrene of other textures) is at least frequently dependent on regurgitant disease of the mitral valve. Four *post-mortem* examinations only were made in these cases; the *green* tint of the softened parts justifies the suspicion that pus may have been present.[2]

"From this rapid survey of the subject of the connection of cardiac and cerebral diseases, what inference can fairly be drawn? None other, I think, than that positive asseverations of the power of heart-disease to generate brain-affections, *as a demonstrated fact*, had best be avoided. On the other hand, I believe that it would be just as unsound to deny totally the existence of any such power on the faith of the numerical comparisons I have just instituted. These are the only comparisons of the sort obtainable at the present hour, but let us not shut our eyes to their serious imperfections. These imperfections are of different kinds. In the first place, some writers are so deeply prejudiced on the question at issue, that their facts cannot be received otherwise than with some quantum of distrust. Read the pages of Andral, and observe how determined he is to find the nexus of an hypertrophied left ventricle, and cerebral hemorrhage; read those of Rochoux, and note the eager partisanship with which he strives to disprove it. Do this, and I feel satisfied you will, with me, see the wisdom of receiving *cum grano salis* their general conclusions. In the second place, some writers put forward cases where no *post-mortem* examination took place; these are, under the circumstances, unfit to form elements in the discussion. Thirdly, who can for a moment suppose it to be at all likely, that all varieties of heart-disease shall have an equal tendency to produce cerebral congestion? That, for example, an hypertrophous left ventricle shall do the same violence to the minute cerebral vessels, if it play through a constricted aortic orifice, as if it play through a perfectly free one. Who can suppose that where the radial pulse is small, feeble, unequal, tremulous from highly developed insufficiency of the mitral valve, the blood shall be propelled with excess of force into the small arteries of the brain? Far from this, the contrary appears so fair an hypothesis, that, as we have just seen, an experienced physician regards such insufficiency as an efficient cause of gangrene-like softening of the brain. The whole clinical history of mitral regurgitant disease points to pulmonary and not to systematic congestion. And *per contra*, the ascertained effects of tricuspid regurgitation give an air of probability to the view that this variety of cardiac imperfection may in reality form the true link between congestive affection of the brain and the heart. Whether this be the fact or not, time will show. Meanwhile, I think there can be no question that it is deeply unsound to club together cases of such opposite functional tendencies as those we have been noticing, and regard them as a single mass producing one single definite effect on the brain. Fourthly, an objection to some of the cases figuring in the returns I have given you lies in the fact, that the respective dates of the cerebral and cardiac diseases have not been clearly made out. And again, fifthly, the subject would require revision, were it only for the change recently effected in our knowledge of the morbid anatomy of enlarged hearts. All enlarged hearts were formerly set down as enlarged by muscular hypertrophy; we now know that the available muscular substance may be less than natural in such hearts, encroached upon and impoverished as it is by accumulating fat. And let us not appeal here to clinical experience to set aside the inference deducible from *post-mortem* investigation. True, the action, felt in the præcordial region, of a fat-infiltrated heart may be agitated and forcible, but violence of *impulsion* does not signify power of *propulsion*."[1]

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[1] One of these counts under the head of hypertrophy also.

[2] Mr. Jordan draws attention to the occurrence of cataract in connection with disease of the mitral and aortic orifices, and with dilatation and fatty disorganization of the heart itself.—Brit. and For. Med. Chir. Review, vol. xix. p. 495.

The correctness of the views here taken seems clearly supported in one aspect by the recent observations of Dr. Kirkes on the detachment of fibrinous coagula from the left side of the heart.

598. *Prognosis.*—Simple hypertrophy, of medium amount, may

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[1] Clinical Lectures, Lancet, vol. i. 1849.

be rendered a very endurable affection by means of regulated diet, moderation in exercise, and general attention to hygienic rules. If the disease be carried to a high point, if it be of considerable amount, and the patient, instead of living according to rule, be forced to work laboriously, and live irregularly, the probable issue is death, through secondary affections, complications and functional derangements, all of which, though originally unconnected with the heart-disease, are rendered more serious by its existence. But pure hypertrophy alone is rarely, and I do not think ever rapidly, the direct cause of death: I cannot call to mind any case, where I have actually known it, and it alone, positively fatal. Dr. Hope, it is true, affirms he has known hypertrophy destroy life in some instances within a year of its commencement (Op. cit., p. 278); still he cites no cases, and appears from the context to have in his mind's eye examples of dilated hypertrophy variously and seriously complicated. Dr. Latham is of opinion, "the heart, *by the simple vehemence of its action*, has the power to kill"—through cephalalgia, insomina, delirium, mania, convulsion, and nervous exhaustion.[1] But by the phrase, "simple vehemence of action," we are not to understand, however this may appear warranted by its terms, such action as an unaided hypertrophous heart in its highest degree can engender. For Dr. Latham gives no proof that the organ possesses any such lethal power, and the only positive case referred to in illustration of the above opinion, is one of hypertrophy and dilatation of the left ventricle following endo-pericarditis and bygone dropsies;—where, too, as no *post-mortem* examination took place, there can be no absolute certainty the encephalon was free from disease. M. Louis calculates the mean duration of hypertrophy of the left ventricle at twenty-eight months, and refers to a certain number of instances in which life was destroyed in from three to seven months. But the evidence is incomplete as to the purity of the hypertrophy [606].

The more dilatation predominates over hypertrophy, the more serious becomes the prognosis—until at last the chances of existence and the quality and amount of suffering become very closely the same as in cases of unmixed, or almost unmixed, dilatation.

599. *Treatment.*—With what hopes may the treatment of pure, or somewhat dilated, hypertrophy of the left ventricle be undertaken? Is the disease curable? Can the nourishment of the heart be not only controlled by artificial means, but reduced below the standard of health? Drs. Latham, Taylor, and Blakiston emphatically deny that art is possessed of any such power; Laennec and Dr. Hope maintain that the feat is easy of accomplishment. But Dr. Hope repudiates the plan recommended by Laennec, and the simple common-sense system lauded by himself fails utterly in effecting the textural changes he ascribes to it. For my own part, I have never

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[1] Diseases of the Heart, vol. ii. p. 338.

known the cure of indubitable hypertrophy proved by physical signs, or by *post-mortem* examination, and hold it unwise to promise any such result from treatment. Even the physical signs may, unless he be very cautious, betray the observer into error in regard of this matter. Thus, not only the impulse may be reduced in force and extent, and the character of the first sound changed, but the area of percussion-dulness lessened, by disgorgement of the right cavities, and yet the heart's actual mass remain precisely as before. All this may sometimes be done, by treatment, in a few days; sometimes not in months—but whether achieved quickly, or slowly, it gives no evidence that hypertrophy has been reduced. On the other hand, it is not difficult to remove or greatly mitigate the symptoms of simple hypertrophy in the majority of cases, and render life not merely tolerable, but comfortable.

600. The theoretical indication is very obviously to tranquillize the heart by diminishing the quantity, without deteriorating the quality, at least materially, of the circulating fluid. For this purpose, occasional bleedings from the arm, to the extent of four, six, or eight ounces at a time, at intervals of from two to six weeks, are recommended by Dr. Hope, in conjunction with a diet mainly consisting of farinacea and vegetables. But even loss of blood to the extent here intended, is more than can be borne by the majority of persons, without slight impoverishment of the fluid; and the least amount of anæmia deeply aggravates the dangers of hypertrophy. Besides, it has appeared to me that over-action of the heart is quite as effectually and as lastingly controlled by very moderate cupping or leeching over the præcordial region, as by the abstraction of a comparatively large quantity of blood from the arm. Four or five leeches, even, will sometimes calm the excitement of a powerful left ventricle, in a state even of somewhat dilated hypertrophy—and this in a well-grown adult.

There is no known drug possessed of the faculty of diminishing the bulk of the heart. Iodide of potassium, pushed even to the production of iodism, fails to exhibit any such power. And though I have an impression derived from tolerably careful watching of some few cases, that this agent may control the disposition to yet further increase, I should, I confess, be puzzled to produce demonstration that such impression is solidly founded. In point of fact, general principles seem the safest guides in the attempt to put a term to increasing bulk of the heart. As rest will waste the muscles of an arm or of a leg, so by analogy must rest, such as can be secured, weaken the nutritive activity of the heart. Quietude physical, emotional, and intellectual is the very first of curative agents for an enlarged heart. To aid in tranquillizing the organ direct cardiac sedatives, hydrocyanic acid, acetate of lead, digitalis, and belladonna (the latter both internally, and in the form of plaster to the surface) must be employed, with occasional intermissions, during the entire treatment of the case. Of all medicines of this class aconite, however, seems the best; the alcoholic extract of the root may be given in doses of one-eighth of a grain with perfect safety in cases of this species; no drug, that I know of, possesses so fully the power of relieving painful sensations and disquietude about the heart.

Purgative medicines, and I believe the saline and aloetic the most appropriate of the class, aid the good effects of rest and small local bleedings. Diuretics are useful, quite independently of the existence of dropsy.

Unless the patient be very highly plethoric, animal food in moderation should be allowed—under all circumstances, indeed, fish may be permitted. Alcoholic fluids, of all kinds, must be avoided; and liquid taken, as a rule, in but small quantity. Walking exercise on a very limited scale is permissible; passive open air exercise more positively advisable.

In treating a case of this kind, the patience of the physician must never fail him: it may require months, nay, years, to produce a favorable effect on the disease—and want of steadiness of purpose and conviction may, in a few days, undo the good accomplished by the efforts of previous weeks. There is one caution to be given to the young practitioner—that he never push any form of treatment to the extent of producing anæmia; the super-addition of anæmia to hypertrophy is that which gives a really ominous character to the latter.

601. II. *Concentric Hypertrophy of the Left Ventricle.*—The discovery that the contraction of the ventricle occurring *in articulo mortis*, increased by cadaveric rigidity,[1] might give to a heart simply hypertrophous the appearance of one concentrically hypertrophous, led, for a time, to the almost complete rejection of concentric hypertrophy as a possible state. But that its occasional existence is matter of fact, would be satisfactorily proved, were this even a solitary example, by a preparation now before me.[2] Here, inclosed by walls equalling very nearly an inch in thickness, lies a left ventricular cavity, which would scarcely give lodgment to a flattened hazel-nut. There appears, besides, in this instance to be very notable relative excess in the thickening of the papillary muscles.

602. The symptoms and signs are those of simple hypertrophy. Theoretically the disturbance of the circulation will be greater, but clinical illustrations of the point are wanting. It has not occurred to me to meet with a pure case of the kind during the life of the sufferer.

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[1] When the spasm occurring *ante mortem* produces the semblance of concentric hypertrophy, it will be found that merely cutting into the tissue of the ventricle causes relaxation; it is unnecessary to have recourse to stretching or maceration. Case of Collins, U. C. H., Males, vol. vi. p. 1, Nov. 1850; death from dysentery.

[2] Univ. Coll. Museum, No. 2140. The reality of concentric hypertrophy has further been proved by M. Déchambre (Gaz. Méd. de Paris, 1844).

603. III. *Hypertrophy of the Right Ventricle.*—Hypertrophy of this compartment, comparatively rare in all forms, is practically unknown except in association with a more or less dilated cavity.

The walls undergo quite as notable thickening, relatively speaking, as those of the left ventricle. In the adult the muscular substance may measure one, or even one inch and a-third in thickness; in a female child, aged ten years, I found it reach a quarter of an inch without, and seven sixteenths of an inch with, the columnæ carneæ.[1]

604. *Physical signs.*—The signs of its dilated hypertrophy are arching of the lower part of the sternum, with greater or less eversion of the ensiform cartilage, and fulness of the epigastrium. The left costal cartilages may be more bulged than the right—a circumstance accordant with the fact, that though the impulse plays very forcibly against the sternum and ensiform cartilage, it inclines, in cases of highly marked right hypertrophy, to direct itself, in consequence of slight displacement of the entire organ, more against the edge of the left, than the right mammary region. Hence it is, that the form of the præcordial region and the site of impulse might betray the observer into the notion that the left, and not the right, ventricle was the seat of hypertrophy. The percussion-dulness may extend considerably beyond the right edge of the sternum; in the child just referred to, it reached an inch outside that edge; at the same time the dulness may be carried unduly to the left also. The distended and enlarged right auricle is the source of much of the percussion-dulness to the right of the sternum.

Dilated hypertrophy of the right ventricle widens the tricuspid orifice in the majority of cases; unless the valve grow in proportion to the widening of the orifice, a regurgitant murmur should, theoretically, occur. Yet, such murmur certainly does not occur by any means constantly, in cases where, *post mortem*, the valve appears physically incompetent. Can this be explained by some constrictive action of the orifice during life which prevents regurgitation? The first sound is duller than natural at the ensiform cartilage; the second fuller, stronger, and more accentuated. Visible jugular pulsation sometimes exists, without the tricuspid valve being demonstrably incompetent.

605. Marked hypertrophy of the right, is so rare without hypertrophy of the left, ventricle, or some form of valvular disease, that it is difficult to give a really clinical transcript of its symptoms. Lividity of the face, and subcutaneous œdema about the face and neck, sometimes exist. Theoretically the lungs must suffer—and hence dyspnœa, engorgement and œdema of the pulmonary parenchyma, bronchitis, pneumonia, tubercles, pulmonary apoplexy, and hæmoptysis, have all been set down by various speculative writers, as dependencies of the affection. Now, of all these alleged symp-

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[1] Kernis, U. C. H., Females, vol. ii. p. 237.

toms, dyspnœa is the solitary one that seems to have thoroughly made out its claim to be so entitled. I believe, however, that engorgement and œdema of the tissue of the lung is not uncommon. Pneumonia, pulmonary apoplexy, and hæmoptysis, I have certainly seen in cases of right ventricular hypertrophy; but in these instances, mitral regurgitation existed also. The notion that right hypertrophy tends to tuberculize the lungs, is not supported by anything I have observed.

The radial pulse, from its being free from all peculiarity of character, affords valuable aid in the diagnosis. Natural in force, while the cardiac action is strong, its state argues against the existence of hypertrophy of the left ventricle, to which some of the local physical signs might otherwise point. The pulmonary artery, of course, bears the force of the hypertrophous ventricle; a fact probably explanatory of the frequency with which, under these circumstances, its coats are atheromatous and calcified.

The difficulty of circulation through the vena cava and jugulars tends to congest the brain venously.

606. According to the results of M. Louis, hypertrophy of the right ventricle runs a mean course of six years and a half; this very remarkable prolongation of life, as compared with the experience of the same observer in regard of the left ventricle, is not easy of comprehension [598].

607. *Hypertrophy of the Auricles—Left Auricles.*—Hypertrophy of the left auricle often accompanies, in variable degrees, constrictive and regurgitant disease of the mitral orifice; but I have never seen it as an isolated state.

608. The symptoms, whatever they happen to be, of these diseased conditions of orifice, are intensified by the overgrowth of the auricle; but I know of none that the hypertrophy can be said specially to produce.

609. Abnormal deficiency of resonance to the left of the sternum about the third and second interspaces, and impulse immediately preceding that of the ventricular systole, are ascertainable physical signs.[1] It is possible, too, that knocking parietal sound may occur.

610. *Right Auricle.*—I have never seen pure hypertrophy of the right auricle, but hypertrophous dilatation, with chronic thickening of the endocardium, is often found, carried to a great extent, in cases of dilatation of the tricuspid orifice.

611. Permanent extension of dulness at the upper right angle of the heart's area will accompany this state, and impulse of præsystolic time will be associated with the venous and other signs of tricuspid regurgitation.[2] As in the case of the fellow-compartment on the opposite side, there are no specially distinctive symptoms.

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[1] F. Smith, U. C. H., Males, vol. v. p. 296, Oct. 1850.

[2] Mann, U. C. H., Females, vol. ii.

DROPSY AND SEROUS FLUX OF CARDIAC ORIGIN.

612. Effusion of the serum of the blood into certain parenchymata and membranous sacs is one of the most important symptoms connected with various affections of the heart; serous flux, though of minor importance, calls for attentive study. And as the precise relationship of both to individual cardiac diseases must still be considered *sub judice*, the subject may, with propriety, be separately examined.

A.—CARDIAC DROPSY.

613. Cardiac dropsies are preceded immediately by venous congestion, itself produced by conditions to be by-and-by inquired into. The fluid composing the effusion, of low specific gravity, contains but a small proportion of albumen, does not coagulate spontaneously, is free from urea, and, as far as I know, any other excrementitious principle, and never forms a blastema apt for evolution.

614. Dropsy, sequential to heart-disease, occurs in the following situations, enumerated in the order of frequency with which they severally suffer; the subcutaneous cellular tissue; the pulmonary parenchyma; the peritoneal and pleural sacs; the pericardium; the cerebral and spinal arachnoid, and sub-arachnoid, spaces; the tunica-vaginalis; the joints; and the eyeball, especially the aqueous chambers. But in the last three situations dropsy is excessively rare.

615. Cardiac anasarca commences almost invariably about the ankles and feet, gradually extending upwards; still I have known it originate about the eyelids, where the kidneys were functionally and texturally healthy; such cases, excessively rare, belong to the class of dilatations with tricuspid regurgitation. When carried to great amount, erythema, erysipelas, and even sloughing are prone to occur; the skin cracks, and through the fissures a fluid at first glutinous, afterwards watery, oozes more or less copiously.

(*a.*) SYSTEMIC FORM.

616. Dilatation was formerly regarded as the condition of the heart mainly inducing the important class of dropsical symptoms. This opinion, discountenanced by M. Bouillaud, who sought to establish valvular obstruction as their sole efficient cause, was restored to favor by Hope and M. Andral—the former of whom, indeed, went the length of teaching that pure hypertrophy, also, was capable of generating dropsy. Of late, Dr. Blakiston has brought together a body of evidence calculated to show that the systemic vessels do not become loaded in cases of dilatation, unless there be co-existent tricuspid regurgitation. Still more recently Dr. H. Douglas has defended the original thesis of our forefathers.

It becomes necessary for me here to express an opinion on this "vexed question" of the mechanism of cardiac dropsy; let me commence by throwing into a series of propositions such inferences as flow directly from facts that have fallen under my own notice.

617. 1. Mitral regurgitation or obstruction, or aortic regurgitation or obstruction, may severally exist, and, for a lengthened period, without systemic dropsy supervening. 2. Mitral regurgitation and aortic regurgitation may coexist for years, and yet no dropsy occur. 3. Both of these propositions hold good, whether notable hypertrophy do, or do not, exist behind the obstruction. 4. Simple hypertrophy of the left ventricle may reach the highest point without systemic congestive effects of any kind arising. Dr. Hope, as is well known, maintained the reverse, holding that pure hypertrophy, if protracted, will produce general dropsy; but he gives no cases demonstrating the fact, and the motives of his belief are, as far as he shows, totally speculative. And *à priori* views are not so completely in his favor as he appears to imagine; when he talks of the "increased force of circulation surmounting the natural tonic power of the capillaries," he forgets that that very tonic power may have increased *pari passu* with, and in consequence of, the growth of the hypertrophy. The question is one of observation; and I have stated what I have actually seen in persons who had not been reduced by treatment to a state of anæmia. 5. Dilated hypertrophy, even, of the left ventricle, may last for years without any such effect, provided the dilatation be not in notable excess. 6. The heart may be in a state of advanced fatty metamorphosis, the pulse feeble and infrequent, the encephalic and respiratory functions exhibit the singular perversions attending a high degree of the disease, the entire organism betray functional languor and inactivity, and yet even the prætibial integuments fail to pit in the least under pressure. 7. Or the heart may be soft and flaccid, and the pulse persistently frequent, feeble, and irregular in force and rhythm, and yet no systemic congestions occur. 8. The natural relationship of width of the arterial orifices, and also of the auriculo-ventricular orifices, may be materially perverted, without the least systemic dropsy arising, until the closing days of life. This is seen, for instance, in cyanotic cases where the aorta and pulmonary artery are transposed in origin, the relationship of the great veins to the two sides being normal.[1] Similar evidence is often afforded by cases of aneurism of the arch of the aorta with an orifice more or less widely dilated, so as to exceed that of the pulmonary to variable amounts. 9. Tricuspid regurgitation, where the right ventricle is in a state of dilated hypertrophy, as shown during life, by swollen and pulsatile jugular veins which fill from below, and as shown after death by actual examination, does not necessarily

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[1] Medico-Chir. Transactions, vol. xxv. p. 1, 1842.

produce dropsy.[1] Besides if tricuspid regurgitation sufficed, unassisted, on mechanical principles to produce systemic dropsy, why should not constriction of the pulmonary orifice, so frequent in cyanosis, habitually engender it?

These propositions are, I believe, incontrovertible; they are the mere general expression of facts which are perpetually occurring. I cannot, then, see how the conclusion is to be avoided, that something beyond and in addition to any one, or any group, of the cardiac conditions referred to, is required in order, as matter of necessity, to entail the occurrence of dropsy. I can scarcely suppose the unwarranted assumption that, were life sufficiently prolonged, those conditions would of themselves suffice for the purpose, would be seriously urged in rejoinder. And again, the existence of some active cause beyond, and independent of, the heart, is further shown by the facts: that there is no direct relationship between the amount of heart disease and of dropsy; that dropsy comes on suddenly sometimes from extraneous causes, the state of the heart remaining, as far as ascertainable, in precisely its previous condition; and that dropsy diminishes and increases, comes and goes, either spontaneously or through the influence of treatment, while the organic changes in the heart remain permanent and unmodified.

We must not, however, run into the opposite and equally erroneous extreme of wholly ignoring the direct influence of organic changes of the heart and its orifices. Two cardiac affections are, as matter of experience, frequently associated with systemic dropsy—namely, dilatation and tricuspid regurgitation. And it is certainly so rare for either of these states to exist for any length of time without the supervention of such dropsy, that any hypothesis, explanatory of cardiac dropsy, must look to these states as forming important links in the chain of causes.

618. A share in the direct mechanism of systemic dropsy being thus conceded to structural change in the heart, the question next arises what is the nature of the influence, independent of that organ, which completes the causation. Local conditions in the heart, we admit, establish a difficulty in the systemic circulation; what influence actually and directly leads to the dropsical exosmosis?

This influence seems a compound of conditions, favorable to transudation of the serosity of the blood, in that fluid itself, in the walls of the capillaries and venous radicles, and in the receiving tissues. *First*, as concerns the blood, the influence of an impoverished state of that fluid is too well known to be for a moment contestable. Experiments on animals prove that if the veins be more or less loaded with water, they yield this in the form of dropsical effusion. The œdema, and sometimes extensive anasarca, of spanæmia; the slight œdema attending the hypinosis of protracted convalescence from various acute diseases; the various dropsies of uræmia—are all illustrations in point. Obviously morbid states of the blood, when of the proper kind, of themselves alone suffice for the production of dropsy; look at the sudden anasarca of acute Bright's disease, or of an acute recrudescence in the chronic affection, while the heart, liver and lungs, may be texturally sound. *Secondly*, it is readily conceivable that the variable density of texture of the walls of the vessels shall promote or restrain the process of filtration. *Thirdly*, cases occasionally present themselves, in which dropsy, supervening from diseased heart, fails to affect portions of the body, noted, under ordinary circumstances, as the earliest and readiest sufferers—for instance, the lower extremities. I have observed this where the legs had been the seat of erysipelas and subcutaneous inflammation prior to the occurrence of the cardiac dropsy: the chronic anatomical changes in the cellular tissue in such a case possibly act as a barrier to its reception of serosity from the vessels.[1]

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[1] W. Hallington, ætat. 53, died at U. C. H., April 7, 1849, with fatty dilated enlargement of the heart (weight 17½ oz.), dilatation of the tricuspid and mitral orifices, and aortic regurgitation, all three carried to a great extent, and yet not a particle even of œdema about the ankles had appeared. The patient had also an aneurism of the aorta, at its bifurcation; but this could not be held to be *preventive* of dropsy.

It may no doubt be argued *à priori* that dilatation of the heart, occurring as a primitive disease through simple weakness, or following actual structural alteration of the texture of the organ, will occasionally prove the efficient cause of dropsy, even admitting the necessity of blood-change just contended for. It may be urged that when the heart is so affected, the necessary *vis à tergo* in the circulation is wanting, and capillary stagnation must ensue—and that this very stagnation, becoming habitual, may modify the qualities of the blood, and impair the nutrition of the walls of the vessels through the strain they suffer. But plausible though this argument be, I believe, as matter of experience, that the necessary change in the composition of the blood is meanwhile really worked out by other and more effective agencies.

Long since, Lower showed that local dropsy might be produced by ligature of veins; and the occurrence of serous effusion from local obstruction of vessels of that class is clinically well known. But here are instances of sudden, and limited obstruction; they are evidently not logically comparable with cases of slow and centric obstruction. And even where the difficulty in the way of the returning blood is purely local, it does not necessarily follow that transudation shall occur from the communicating venous radicles, although the evidences of changed condition of those vessels be structurally obvious. Thus in cases of aortic aneurism, pressing on one or both innominate veins, the capillaries of the base of the neck may dilate in such manner, as to produce notable general swelling at the spot, yet not a particle of œdema be discernible.

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[1] Clin. Lect., case of Hope, Lancet, 1849, vol. i. p. 442. Robertson, U. C. H., Females, vol. viii. p. 295.

619. *Treatment.*—The treatment of cardiac systemic dropsy must vary somewhat with the precise conditions of the heart; but there are some points of general applicability which may here be set down. There is no period of valvular disease at which the removal of dropsy may not be accomplished; but when the powers of the patient are seriously enfeebled, great caution, as regards the rapidity of that removal, is called for.

The dropsies of heart-disease are very rarely attended with true plethora, and hence on that score very rarely call for venesection. But, when a clogged condition of the heart's cavities coexists with pulmonary congestion and anasarca of more or less acute course, and the general vigor is as yet unimpaired to any serious degree, the abstraction of a small quantity of blood, either by venesection or cupping of the chest, has appeared to me to facilitate the action of hydragogue remedies. That diminished fulness of the vascular system promotes absorptive power, is well known; but even where the quantity of blood abstracted scarcely authorizes us in resorting to this explanation, the result is beneficial.

Of hydragogue purgatives, peculiarly valuable from their certainty of action, elaterium,[1] gamboge, bitartrate of potass, and the pulvis jalapæ compositus are the most valuable of the class. Stimulants may be requisite during the action of these medicines; and their action may be so rapid, that the mere loss of fluid shall weaken vitally and mechanically. An ounce and a half of the bitartrate of potass may be given with perfect safety, freely diluted and combined with carminative tinctures—smaller doses having first been employed.

Diuretics, uncertain in their action, are often rendered much more active by preliminary cupping or dry-cupping of the renal regions. It will be necessary to vary the combination from time to time, and the list for selection is fortunately a full one—comprising the acetate, nitrate and bitartrate of potass, the iodide and bromide of potassium,[2] nitric ether, the spirits and infusion of juniper, squill, colchicum, decoction of scoparium and of chimaphila, and if the heart's strength be not seriously impaired, infusion of digitalis, or if the liver be at all engorged, the extract and decoction of taraxacum. Hydragogue purgatives often promote the action of more direct diuretics; and small doses of blue pill, occasionally at bed-time, are very useful adjuvants.

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[1] The formula I employ is as follows: ℞ Extract. Elaterii, gr. $\frac{1}{8}$—gr. $\frac{1}{2}$; Creasotonis, gt. i.; Extract. Hyoscy., gr. ii.; pro pill., i. The inequality of strength of the extract, as supplied by various druggists, renders it prudent to commence with a very small dose: the greater certainty of the strength of the alkaloid, momordicine, will probably lead one day to its being generally employed, though with a purgative of such intense activity, and, at the same time, occasionally depressing character, the greatest caution must, of course, be observed.

[2] The iodo-bromuretted waters of Adelheidsquelle, Kreuznach, and Woodhall, act diuretically in some cases.

Diaphoretic drugs are of little utility; but the free diaphoresis produced by the vapor, or hot-air bath, is sometimes strikingly beneficial.

If there be any tendency to bronchitis, bronchial discharge should be encouraged by the free use of expectorants.

Removal of anasarcous fluid by mechanical means becomes necessary, if medicines fail in controlling its increase; and if inflammatory changes in the skin appear imminent. Scarification is hazardous, being not unoften followed by erysipelas; while acupuncturation at the upper and inner parts of the thighs, though a slower process, is not attended with the same danger; there is even some advantage in the slowness of discharge; too rapid evacuation has been followed by great depression. On the other hand, should discharge have commenced, either through spontaneous fissures or artificial punctures, it is extremely dangerous to arrest the flow by healing the openings; I have known death rapidly ensue from dyspnœa and thoracic dropsy under the circumstances.

The patient's strength must be supported by food containing much nutriment in a small compass; and, though thirst torment him, he must refrain from fluids as far as possible. Stimulants may be required; and hollands or gin have both popular prejudice and medical experience in their favor under the circumstances.

(b.) VISCERAL AND INTRA-SEROUS FORM.

620. *Pulmonary œdema* is chiefly connected with disease, constrictive or regurgitant, of the mitral orifice, sometimes with dilatation of the left ventricle, very rarely with hypertrophy of the right. *Ascites* arises especially from general dilatation of the heart and tricuspid regurgitation: and these are the conditions mainly observed as antecedents to all the other varieties of dropsy enumerated. Of the mechanism of hydropericardium I have already sufficiently spoken.

B.—CARDIAC FLUX.

621. (*a.*) The intestines become the seat of serous flux in some rare instances of certain forms of heart disease. These forms are general dilatation and tricuspid insufficiency. Watery diarrhœa thus induced may be wholly spontaneous, or the original excitant of the discharge may have been some hydragogue purgative, the influence of which holds on. Unless excessive, flux of this kind is rather to be encouraged than otherwise, probably saving the patient from dropsy of the areolar tissue or of the serous sac. But as far as my observation goes it rarely occurs in the purely spontaneous form; when it does so, its utility is much greater than when excited medicinally.

622. (*b.*) I believe that in some of those cases where vomiting forms so obstinate a symptom of chronic heart-disease, the immediate cause of the nausea is the presence of an irritating watery secretion from the gastric surface. It is never very copious.

623. (*c.*) Watery flux from the kidneys, though on the whole not very rare, is uncertain in its occurrence, doubtful in its mechanism, and cannot be said to belong to any particular cardiac affection. For my own part I have seen passing attacks of hydruria more frequently in connection with different forms of dynamic disturbance than with any specific organic disease. It is not very uncommon for fits of palpitation, purely functional, to terminate by, or with, a copious discharge of aqueous urine; so, too, anginal and pseudo-anginal seizures, or passing fits of mere paræsthesia of the cardiac nerves, may be followed by hydruria. I know a case of the latter class in which two and even three quarts of such urine were occasionally discharged in the twenty-four hours after the seizure, the patient being habitually remarkable for the small quantity of his renal secretion. Now, though the doctrine of eccentric pressure on the Malpighian capillaries, already referred to [593, *h*], might with some plausibility be adopted in the case of hydruria sequential to grave palpitation, I do not see the smallest *locus standi* for such explanation, when the discharge follows simple paræsthesia. Here the rationale is the same as in the case of hysterical diuresis; and so long as the immediate mechanism of the latter continues in its present obscurity, we must remain in ignorance of that of the former.

624. Albuminuria occurs every now and then in cases of cardiac disease, especially of dilatation and tricuspid insufficiency, the tendency of which is to congest the viscera. The cortical and tubular substances remain texturally sound.[1] The characters of this form of albuminous flux are as follows: it is a passing phenomenon, sometimes disappearing spontaneously, sometimes yielding to treatment; the albumen is always small in amount; the specific gravity of the fluid never falls very low, its color remains unaffected and its smell wholly free from that whey-like odor to which I long since drew attention as notably significant of "Bright's disease."[2] A few casts of the tubules occasionally appear.[3]

IV.—DILATATION OF THE HEART.

625. By dilatation of the heart, is understood that state in which the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms: *hypertrophous dilatation*, where dilatation predominates, but the walls are some-

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[1] G. Smith, U. C. H., Males, vol. xi. p. 196; also Couch, U. C. H., Females, vol. vii. pp. 341—345. But examples of the fact are of frequent occurrence.

[2] Clin. Lect., Lancet, 1849, vol. i.

[3] Just as they do in pyelitis, in jaundice (Scott, U. C. H., Females, vol. v. p 93), and probably in a variety of other affections.

what thicker than in health; *simple dilatation*, where the walls are of such thickness as would be normal, had the capacity remained unchanged; and *attenuated dilatation*, in which the walls are distinctly thinner than in health—to such a degree that the thickness of the wall of the left ventricle may, in some points, positively not reach one line. Clinically, as well as anatomically, the characters of dilatation are obvious in proportion to the excess of capacity over thickness; and the description of the disease about to be given must be understood to refer to cases in which such excess is prominently marked.

626. Dilatation, carried to serious amount, usually involves both ventricles; one, however, to a greater extent than the other. The disease is not uncommonly limited to the right, very rarely to the left, ventricle. The form of the dilated ventricle becomes globular, and the apex of the heart so rounded off, that it may with difficulty be detected. The transverse measurement of the organ undergoes much greater increase than the vertical. In rare instances the auricles are predominantly affected.

Dilatation of the orifices only occurs, as a rule, where both auricle and ventricle on the same side are implicated: insufficiency of either auriculo-ventricular valve may thus be produced independently of structural disease or even dynamic imperfection in itself. Dilatation, especially when pushed to extremes, notably impairs the activity of the blood's movement through the heart—hence follows a tendency of variable force to accumulation of blood in the cavities. The physicial signs of such accumulation may occasionally be caught during life, and after death grumous coagula are commonly found—the tendency being sometimes, though rarely, counteracted by the actual mode of dissolution.

The tissue of the dilated walls is more or less soft and flabby—and if this deficiency of firmness be disproportionately marked at any particular spot, the ventricle may there yield, undergo pouching, and a true aneurism of the heart ensue. The color may be deep brownish red, anæmically pale, or mottled yellowish brown. Microscopically, the fibre is deficient in striated character, and granular looking; fat, extra or intra-sarcolemmous, may exist; and in some cases the appearances of adventitious cellulo-fibroid texture are traceable, the muscular substance being cirrhosed and atrophous.

627. *Causes.*—If any one of the affections of the orifices of the heart, tabulated in a former place as efficient causes of hypertrophy [589], act upon a heart of weak resistant power, and disposed to an under-par state of nutrition, the result of the inward eccentric pressure of the blood on the endocardial surface will be dilatation.

But no constant ratio holds between the amount of dilatation and the apparent valvular difficulty or difficulties: and in certain cases there is found positively no statical mischief; and, as far as can be judged, no dynamic disturbance has existed during life about the orifices. Hence it is supposed that weakness of the heart's fibre, pure or independent of definite structural change, renders the walls of the organ incapable of resisting the eccentric strain arising under the ordinary effort of the circulation.

But, there is, besides, the influence of mechanical difficulty beyond the heart itself to be considered. Does aneurism of the aorta entail dilatation of the left heart? We have no proof that it does. I know, as matter of observed fact, that not the smallest increase of area or of bulk of the left ventricle may exist, even when a very large sac originates just above the sigmoid valves; and hence feel disposed to believe that dilated hypertrophy and aneurism, when found together, are mainly related as joint results of a common cause.

Persistent capillary obstruction in various organs is ordinarily supposed to dilate the heart in the course of time. The arguments used in considering the power of renal obstruction to produce hypertrophy will apply in the case of dilatation, quoad the mechanics of the question [596]. The influence of all obstructive diseases of the lung of asphyxiating character in dilating the right, and eventually the left, heart, has long been known. Mere capillary obstruction and destruction will not suffice for the purpose; for as a rule the heart, instead of rising above, falls below, the par of healthy bulk in phthisis. A probable enough explanation is that in phthisis the mass of the circulating blood undergoes gradual decrease, proportional as it were to the capillary destruction within the lung. Again, it may be, that, as Dr. Gairdner suggests, enlargement of the heart takes place in collapse of the lung and emphysema, "because the expansion of the thorax in inspiration tends constantly to overload the heart."[1]

There still remain cases wherein dilatation, inexplicable on any of the theories now reviewed, appears traceable to some intrinsic fault in the heart itself, the nature of which remains unrevealed.

628. It seems somewhat like "slaying the slain," to denounce the doctrine of absolute mutual repulsion between cardiac diseases, such as dilatation, attended with venosity of the blood and active tuberculization. A single fact, elsewhere recorded, would suffice to show the theory cannot stand.[2]

Cases occasionally occur, telling both for and against the theory. Thus a patient may be tuberculous, and then become the subject of dilatation and tricuspid regurgitation, while pre-existing tubercle either becomes quiescent or undergoes retrograde changes; and yet the death of such a patient, whose life holds on at the time despite a persistent partial asphyxia, maintained by the heart disease, may be in fact eventually due to the acute development of gray granulations.[3]

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[1] Brit. and For. Med.-Chir. Rev., July, 1853.

[2] Diseases of the Lungs, 3d Am. ed., p. 409; also Crocott, U. C. H., Males, vol. xviii. p. 113.

[3] Clin. Lect., case of Hishin, Med. Times, February, 1857.

629. In order as far as possible to insure precision, it seems advisable to consider apart, dilatation involving the heart as a whole, and dilatation of particular cavities. The student will, however, bear in mind that the latter cases are rare, and of relatively small practical importance.

A.—GENERAL DILATATION.

630. *Physical signs.*—The apex-beat is very indistinctly visible, or, if the patient be full in person, actually invisible; when seen, it may fall nearer the sternum than the natural point of the apex-beat, in consequence of the rounded-off form of the heart annulling, or lessening the clearness of definition of the true apex; if the dilatation be considerable, the apex strikes the surface *below* the fifth interspace. The impulse, whether more extended than natural, or limited to the fourth and fifth interspaces, frequently exhibits the quasi-undulatory character. There is no prominence of the cardiac region.

As felt by the hand, the shock is feeble in proportion to the purity of the dilatation: the apex-beat, though visible, and though movement be nowhere else visible, frequently cannot be distinguished by the finger from the impulse of the ventricles generally—an evidence of extreme motor feebleness. The impulse, non-vibratile, either consists of a short feeble slap, followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion: the force of successive beats is unequal; their rhythm may be irregular to a slight or to the very highest degree: there is a want of perfect uniformity in the point of the surface struck by the heart in the successive beats of a series, quite independently of the influence of respiration: palpitation may notably increase the force of impulse, though it often does so but very slightly.

The intensity of percussion-dulness in the superficial cardiac region is not, as it probably is in hypertrophy, increased; and in cases of attenuated dilatation the parietal resistance, it is averred, may be less marked even than in the state of health. Both these signs are, to say the least, difficult of detection and insignificant in value. The phenomenon of horizontal conduction of percussion-sound [99] must always interfere with both. The areas of dulness, deep and superficial, are both widened: the former inclines to squareness of outline, the transverse diameter of the heart having proportionally undergone a notably greater increase than the vertical.

The systolic sound varies in characters according as the cardiac tissue is firm or soft—a variation which is intelligible whether muscular action be, or be not, accepted as an actual part-cause of the sound. In the first place, if the fibre be firm and fairly nourished, the heart's systolic sound, both at the base and apex-region, is, generally speaking, though short and abrupt, full-toned and of higher pitch than in health. It is superficial, appearing to be produced close under the spot of chest surface auscultated; its maximum point is generally slightly lowered. The diastolic successor, is not specially affected. Both sounds, as heard at the top of the sternum, bear the natural relationship, in point of intensity and clearness, to those audible over the heart itself—a circumstance which occasionally helps the observer signally in excluding hydropericardium, as a possibly existent state. In the second place, if, on the contrary, the dilated ventricle be soft, flabby, or fatty, the first sound may be very weak, faint, toneless, and of high pitch; and the second so feeble as to be inaudible at the apex, especially if the heart's rhythm be markedly irregular. So, too, upon the quality and mass of the heart's texture will depend the extent to which the sounds are transmitted through and over the chest; if the dilated ventricles be well nourished, and *à fortiori* at all hypertrophous, the sounds, approaching in attributes those of hypertrophy with dilatation, will be widely diffused, and clearly audible in the right back; if, on the contrary, the dilated heart be a soft, flabby one, the area over which its sounds are audible may be extremely limited. A slapping noise, the intensity of which may be increased by causing the patient to bend forwards, is sometimes produced by the collision of the heart against the side.

Dilatation is not habitually productive of any intra-cardiac murmur, unless the morbid state has proceeded to such lengths as to render certain valves incompetent to close their orifices; this may occur with the tricuspid, more rarely with the mitral, and still more rarely with the aortic valves. The murmurs thus engendered are, of course, always regurgitant; but they do not always occur when their physical cause, incompetency of the valves, actually exists, inasmuch as the strength of the ventricles is sometimes incapable either of giving the necessary impetus to the regurgitating stream, or of calling forth such energy of its own systolic reaction on the part of the aorta, as is required for the production of murmur. It is also possible that dilatation, by giving an unnatural direction to the blood-current, may engender murmur [188]. Are reduplications common when the ventricles are unequally dilated? I think so; but my number of observations on the point is too small to justify a positive assertion.

631. *Symptoms.*—Few affections vary more notably in the intensity, few agree more completely in the character, of their local and general symptoms, than general dilatation of the heart. To describe all the degrees clinically observable, modified too as they are by the idiosyncrasy of the individual and by the manner of complication with changes structural or functional of other parts, would prove an endless task; it will suffice to place before the reader a picture of the maximum amount of suffering the disease entails.

And it is important to remember that cases in which dilatation of the heart exist in an isolated state symptomatically are rare: in the first place such dilatation, as an actual pathological state, is

(*d.*) Anorexia and nausea may be habitual. The latter, indeed, occasionally becomes the dominant source of suffering—the sense of nausea scarcely ever ceasing, while the simplest food in the smallest quantities is invariably rejected. In this state there may be no biliary derangement, and the epigastric region be perfectly free from pain or tenderness. The mechanism of the symptom probably varies in different cases—sometimes depending on mechanical congestion of the gastric mucous membrane, sometimes on mechanical pressure by the heart from above, sometimes on peculiar secretion [622], and sometimes on reflex irritation of the vagus nerve. I have occasionally known the symptom disappear spontaneously after having resisted all kinds of treatment for weeks together. The tongue (unless some intercurrent state exist to modify its characters) is broad, full, not always pitted at the edges, however, and of dark purplish ground—the fauces venously injected. Hemorrhage may occur from various parts of the alimentary mucous tract; I have seen this in the case of the bowels, when the dilatation alone seemed its direct cause, but do not feel positive that hemorrhage from the stomach has occurred under my observation, unless there were intermediate high congestion of the liver—the hepatic system may act as a sort of safety-valve for the stomach, until itself grows overloaded. Hæmorrhoids are not so common as might be expected—the bowels habitually constipated, or alternately relaxed and confined—the discharges dark. The liver is more or less congested: the hepatic system suffers first, the portal secondly; unless both are implicated, the size of the organ is not sufficiently increased to affect the results of percussion notably: the texture of the lobules may remain perfectly sound. Positive jaundice does not occur from this state of the liver alone; but I have seen a faint yellowish tint during life, where death disclosed nothing but hepatic congestion to account for it. Ascites follows on anasarca; I have no experience of the peritoneal dropsy as a precursor of the cellular.

(*e.*) Venous stagnation, with its consequences, occurs in the lungs. —Here may be enumerated oppressed or sighing breathing, and eventually orthopnœa, complete and habitual, with occasional asthmatic paroxysms. The respiration may actually stop when the patient dozes—as if the influence of the will were necessary to sustain the act. This is probably a symptom of fatal augury—as it certainly proved in the few cases I have observed it. In addition cough, dry, harassing, and even convulsive, or accompanied with expectoration, serous, rarely frothy, or stained or streaked with blood, or mixed with a little blood, fluid and dark, or in pellets, increases the distress. The physical signs of bronchitis, or of pulmonary congestion, irregularly disseminated; though most marked at the posterior bases, of pulmonary œdema, or of pulmonary apoplexy, may be found—actual hæmoptysis to large amount may accompany the latter state quite independently of mitral disease.[1] In rare instances considerable hæmoptysis occurs, where the anatomical conditions of pulmonary apoplexy are not to be found after death.[2].

(*f.*) The organs of circulation themselves furnish the following symptoms. Feeble, fluttering, distressing palpitation, increased by the slightest movement, occurring from some obvious cause, or as frequently without apparent excitement, is a standing source of misery to those sufferers. So, too, is uneasiness in the cardiac region, of characters most difficult to describe—of an intensity varying between a mere sensation which constantly reminds patients that, as they often express themselves, "they have a heart," (the mildest form of cardiac paræsthesia) and the agony of angina—paroxysms of which may actually occur and put an end to existence. Tenderness on pressure may be discovered over the heart in some rare cases. The pulse is either small and feeble, and abnormally posterior in time to the ventricular systole, but regular; or it is narrow, feeble, fluttering, and irregular both in force and rhythm. The latter state is either limited to the periods of palpitation, or, if constant, is indicative generally of softness of the heart's texture. True intermittence is rare. The superficial pulses are not visible. Faintness, occasionally lapsing into actual syncope, occurs from time to time.

(*g.*) Swelling of the abdominal lymphatic glands is said sometimes to take place: I have never seen it—possibly because sufficient search was not made.

(*h.*) The kidneys are passively and mechanically congested like the lungs and liver; the renal regions may be tender on deep pressure posteriorly; but I have never known the organs sufficiently increased in bulk to cause any positive extension of the area of their posterior lumbar percussion-dulness. The urine is small in quantity, high-colored, loaded or not with lithates. Albuminuria may occur from mere renal congestion—the urine possessing the characters already described under the head of cardiac flux [623]: this symptom often disappears spontaneously, and may almost always be quickly removed by certain measures directed to the kidneys, especially cupping or dry-cupping. In rare instances spontaneous hydruria occurs temporarily; I have known most copious diuresis of the kind preceded by violent rigors, for which no cause could be detected.

(*i.*) Sexual inclination, though weakened materially, is not annulled—rupture of a dilated heart has occurred *in actu coitûs*. It is said that uterine hemorrhage is sometimes traceable solely to dilatation of the heart: the statement, unconfirmed by any cases that have fallen under my notice, seems to stand in need of corroboration. I certainly have, nevertheless, seen some instances of profuse menstruation in females of an age more or less advanced beyond thirty—and in whom coexisting mechanical congestion of both liver and kidneys made it probable enough, that a similar condition of the uterus might have played a part in increasing above par the quantity of discharge through the vagina.

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[1] Fosbury, U. C. H., Males, vol. x. p. 133; Whittaker, U. C. H., Males, vol. xviii. p. 85.

[2] The minute branches of the pulmonary artery are then probably themselves diseased. Vide Dis. of the Lungs, 3d Am. edit., pp. 324, 327.

(*k.*) More or less congestion of the encephalon can scarcely be escaped, where the face exhibits marked indications of stagnating circulation. The dull cephalalgia, inability to exercise the mind, heaviness, torpor, somnolence, semi-coma, and finally, coma, that mark the progress of these cases, are thus explicable. Cerebral hemorrhage and acute softening both stand in undetermined relationship to dilatation. No matter what the amount of mechanical congestion may have been about the face and head, it has never occurred to me to witness convulsive seizures, even distantly allied in character to the epileptic, as results of that congestion.

(*l.*) Congestion of the spinal cord may be the cause of certain reflex phenomena, spasmodic cough, startings from slumber with affright, and convulsive actions of short duration on waking from sleep.

(*m.*) Muscæ volitantes, luminous vision, fulness, wateriness, and injection of the eyeballs; tinnitus aurium, dulness of hearing; with deficiency of smell, and occasional epistaxis, from congestion of the Schneiderian membrane; all indicate participation on the part of the organs of sense.

Taking the systemic and the pulmonary obstructions as two separate classes, the former have occurred under my observation almost invariably before the latter: Dr. Hope taught the converse, but it appears to me, on theoretical grounds.

633. *Diagnosis.*—The diagnosis of dilatation of the heart turns mainly on the following points:—Weak action, quasi-undulatory impulse, indistinctness of the apex-beat; increased area, with squareness of outline, of the percussion-dulness, the whole not lowered in proportion to its width; heightened pitch, shortness, abruptness of the systolic sound, or great deficiency of tone in certain cases, with prolongation of the post-systolic silence; peculiar characters of the pulse; and signs and symptoms of systemic and pulmonary obstruction and congestion.

Simple hypertrophy is distinguished from dilatation by the forcible action of the heart; by the præcordial bulging; by its distinctly localized thrusting impulse; by the heart being lowered in proportion to the general area of dulness; by the muffled dulness and prolongation of the first sound and shortness of the first silence; by the characters of the pulse; and by the systemic signs being those rather of an excited than of a stagnating circulation. Every one of these characters differs more or less from those of dilatation.—*Eccentric hypertrophy*, also, has its forcible action, præcordial arching, and distinct apex-beat; the heart is lowered in the proportion of its bulk; the systolic sound is loud and powerful; and the pulse differs from that of dilatation. The only difficulty comes of the fact that, if the cavities be more increased in capacity than the walls in thickness, the systemic and pulmonary signs may incline to those of pure dilatation.—*Mitral regurgitant* disease will, under ordinary circumstances, be distinguished from dilatation by its special systolic murmur at the left apex, and by the more forcible impulse: for some amount of hypertrophy in the vast majority of cases ensues on the valvular disease. Still there are two kinds of possible fallacy. On the one hand pure dilatation may be attended with the murmer indicative of regurgitant disease of the mitral valves, though those valves be sound. On the other hand, organic mitral regurgitation may, *pro tempore*, be murmurless. Now, the first difficulty will occur where the orifice is so much dilated that its valve, though perfectly sound, has ceased to be of sufficient size for purposes of complete closure. There are no possible means of distinguishing such regurgitation from that induced by actual disease of the valve; happily, the phenomenon is very rare; and, as the other characters of dilatation must be present in a high degree, the treatment will not err. The second difficulty—that of murmurless organic regurgitation—will arise from feebleness of the backward current, itself depending on temporary general collapse, or weakness limited to the heart; in a short while, the heart's vigor having improved, mitral murmur becomes audible.—Dilatation differs from *chronic pericardial effusion* by the square outline of its dulness; by the distance from the clavicle to the spot of the apex-beat being natural or increased; by the heart's sounds being superficial, and of about the same strength at the præcordial region as at the top of the sternum; by the dulness not extending above the third rib; and by the total absence of friction-sound, no matter in what position the patient be placed.

634. *Prognosis.*—Under all circumstances a most serious disease, the danger of dilatation increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months.

635. *Treatment.*—Dilatation of indubitable existence is not removable by treatment: those doubtful cases of the disease, which Dr. Hope professed to cure, bear no claim to scientific recognition. But if it be not, in the highest sense, a curable affection—though art cannot remove dilatation—art can render dilatation bearable, and even unfelt. In conducting the treatment, the essential clinical element of the disease, weakness (whether dilatation be primary or secondary), must be constantly held in view, all debilitating mea-

sures systematically avoided, and the effort made to improve the heart's tone without exciting its irritability.

Unprovided, as we are, with any medicine exercising a specific action on the tone of the heart's fibre, ordinary general tonics must be resorted to. Bitters, mineral acids, and preparations of iron, in doses and combinations modified according to circumstances, supply the groundwork of medicinal treatment: it is of course to be understood that no positive contra-indication to the use of these agents exists beyond the heart. Opium or belladonna, internally and externally, tranquillize excited action better and more safely than other sedatives.[1] Aconite may be given in very small doses, on urgent occasions, but by no means employed habitually. The exhibition of digitalis requires the utmost caution: slackening the circulation, as it does, it promotes either coagulation within the heart, or, in a less degree of its action, accumulation of blood in the cavities, whereby they may be still further passively dilated. If the power of the ventricles be seriously impaired, digitalis cannot be given without excessive risk, and had much better be altogether avoided. Should the heart be nervously excited, the various antispasmodics are indicated. The inhalation of chloroform or ether ought not, under any circumstances, or for any object, to be lightly permitted.

The condition of the chylopoietic viscera must be carefully watched—due action from the bowels insured daily, without weakening purgation (aloetic medicines are the best), and the action of the liver, if this be sluggish, promoted by taraxacum and an occasional mercurial aperient.

The patient should lead a tranquil life, avoiding all excitement, but mixing in cheerful society. His exercise should be moderate, always considerably within fatigue. It does not appear to me that because a patient with dilated heart occasionally feels better able to walk after some minutes' effort, than he did at the actual start, therefore it is advisable to push pedestrian exercise as far as it can be endured. The natural history of angina would suffice to prove such strained work to be wrong. And, in truth, I cannot help thinking that error is frequently committed in the attempt to force patients, laboring under various diseases, to *walk*, *walk*, *walk*, in spite, often, of their own convictions that each additional walk has helped them on towards that bourne whence no walker returns. I do not refer, merely, to the bygone well-known practice in a central provincial town—but to the too indiscriminate recommendation of toilsome foot-exercise by many of the profession generally. I have known a man laboring under chronic nephritis, with alkaline urine and the general cachectic condition of that disease, directed to perform such pedestrian feats, by way of recovering his health, as he himself well knew would, on the very first day, have brought on such an acute attack, as probably must have confined him to bed or a sofa for a month. I entertain not a doubt that the lives of sufferers from cardiac disease are often put in jeopardy by the effort to exercise beyond their powers. Because, in the *physiological* order of things, it is good to walk, the inference is at once drawn that it is universally good in the *pathological* order of things. But, in point of fact, each disease has its own appropriate kind and quantity of exercise—and that kind and that quantity are, as all truths in therapeutics, to be learned in each instance by *experience* alone. By the way, the Leamington practice once more tells the tale, "there is nothing new under the sun!" The ancient gymnastic physicians outdid their followers of the 19th century. Plato states that Herodicus prescribed a pedestrian excursion from Athens to Megara and back, a distance of 360 stadia, or upwards of 40 miles![1]

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[1] The influences of belladonna and opium are in some points of view so unlike, so opposed, indeed, that possibly the states of heart and circulation relieved by each, are not so really similar as they appear. See a paper by Dr. T. Anderson, on the Effects of Belladonna in Poisoning by Opium.

Very gentle use of light dumb-bells, by promoting the circulation through the upper extremities, seems to act beneficially; but caution is requisite in permitting this.

A good nourishing animal diet, of plain, easily digestible character, is that fittest for these patients. Much drink of any kind is to be avoided; bitter beer in moderation is allowable at dinner, or, if the patient have been accustomed to take these, a glass, or at most two, of port or sherry; dandelion coffee is a good material for breakfast. A dry, bracing air, as a rule, agrees best. Particular circumstances, it is true, may require the patient to live in one of the very opposite characters; but, though a relaxing atmosphere may relieve an accompanying dry bronchitis, it unquestionably tends to depress still further the heart's energy. The use of flannel next the skin is indispensable; the shower-bath may be cautiously tried, and continued or not according to its visible effects—effects which, it has appeared to me, are, in all diseases, much under the influence, first, of idiosyncrasy, and, secondly, of the patient's previous habits in respect of ablutions. Tepid salt-water bathing, or even, when the affection is not far advanced, quiet immersion in the open sea, is useful; swimming exercise, it is scarcely necessary to say, should be absolutely avoided.

Paroxysms of dyspnœa may be relieved by hydrocyanic acid, cannabis indica, ether, and liquor opii sedativus, and the ethereal tincture of lobelia inflata; by dry-cupping, or the application of three or four leeches to the præcordial region, especially if there be palpitation; or by the use of Junod's exhausting apparatus. Tendency to arrest of respiration during dozy sleep may be averted by the use of a gentle electro-galvanic current from the nucha to the epigastrium. I have tried this with some benefit. If disturbed cardiac action be traceable to hysteria, the usual remedies for that state should be given; if to flatulence, carminatives sometimes tranquillize the organ almost instantly. Pulmonary congestion and sub-inflammation require dry-cupping, mustard poultices, flying blisters; and, as they are rarely active, squill and ammonia or senega internally. Should actual pulmonary inflammation occur, antimony must be employed with great caution: it is easier to depress the vital powers than to raise them. Systemic dropsies are removable by the methods elsewhere described [619]. The fluid should not be too rapidly drawn off, supposing surgical influence becomes requisite, as the sudden loss of support on the part of the vessels may induce prostration of strength, from which the patient cannot be recovered: this proposition holds good in respect of acupuncturation of the limbs as well as of paracentesis abdominis. Congestive enlargement of the liver is rapidly reducible in some patients by dry-cupping over the organ. Even the rest from toil and general comfort of an hospital in patients previously suffering from privation of all kinds, will occasionally free the cardiac circulation sufficiently in a few days, to lessen appreciably the antero-posterior and vertical measurements of the liver through disgorgement.

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[1] Cabanis, Medical Sketches, Tr. by Henderson, p. 406.

B.—DILATATION OF PARTICULAR CAVITIES.

RIGHT VENTRICLE.

636. *Physical signs.*—Dilatation of the right ventricle in particular is signified by extension of percussion-dulness to the right, by excess of epigastric pulsation, by jugular turgescence, and, if there be either tricuspid regurgitation or some muscular strength in the wall of the ventricle, by jugular pulsation of cardiac rhythm and refilling of the emptied jugular veins from below. Further, the chances of coagulation of the blood occurring within the cavity, especially when the walls are notably weak, are always to be borne in mind; systolic murmur, following the course of the pulmonary artery, may thus be generated.

637. *Symptoms.*—It is difficult to affix to dilatation of this ventricle any symptoms that may peculiarly depend upon it—so frequently does some amount of dilatation of the left ventricle or some form of valvular impediment exist to complicate the problem. Theoretically, the signs of deficient oxygenation of the blood will be marked in proportion to the amount of pure or attenuated dilatation of this ventricle—as will, also, the congestive influences on the brain and abdominal viscera. Whatever argument may be urged as to the power of unassisted hypertrophy of the right ventricle to produce hæmoptysis, it is very positive dilatation of the cavity will exercise no such influence.

AURICLES.

638. Direct physical evidence of dilatation of either auricle is only to be had by percussion in the natural sites of those cavities.

When the jugular veins are permanently dilated and knotty, the existence of dilatation of the right auricle is inferrible, almost as matter of necessity; when the evidences of mitral disease have been unmistakably developed for any length of time, a similar inference applies to the left auricle.

It would appear from a case observed by Dr. Markham,[1] that in cases of extensive extreme attenuated dilatation of the heart especially affecting both auricles, thrill, loud murmur, and pulsation, synchronous with the auricular diastole, may occur in the fifth interspace considerably to the right of the sternum—physical signs which the observer suggests may have depended on the rush of blood from the venæ cavæ into the auricle.

639. No special symptom can be assigned to dilatation of one or the other auricle. But the influence a dilated auricle exercises in increasing the amount of disturbance caused by augmented capacity of the connected ventricle is very manifest.

V.—Alterations of Consistence.

A.—SOFTENING.

640. *Conditions of origin.*—Diminished consistence of the muscular structure of the heart forms an important physical element of a variety of complex morbid states of the organ. Thus the fibre loses consistence in carditis, in pericarditis, in endocarditis (at least in the strata adjoining the inflamed membrane), in hemorrhagic infiltration, in œdema, in fatty infiltration and in fatty metamorphosis, in pure dilatation, and in certain forms of atrophy. So, too, the tissue loses firmness in certain diathetic diseases of the blood-class, in which the quality of the fibrin, whatever may be said of its quantity, undergoes impairment—as, for instance, in cyanosis, scurvy, purpura, leucohæmia, glycohæmia, and acute phthisis. Lastly, more or less marked softness of the heart, apparently arising out of weakened innervation, as well as the altered quality of the circulating blood, occurs in variola, scarlatina, typhoid (Peyerian), and especially typhus fevers.

Now in all these instances, softening constitutes an accident of the primary affection; but there are other cases in which the heart notably loses its consistence, without there being any prominent blood disease, or any obvious affection of the organ itself, except the deficiency of firmness. To the latter cases alone it is proposed at present to refer: they are, perhaps, more uniformly connected with fatty alteration, than has at the present day been demonstrated.

641. *Physical signs.*—The impulse of a heart, thus variably reduced in consistence, may be persistently invisible, or visible with some and invisible with other beats, and occasionally somewhat undulatory. If any single beat be of considerable force, the left

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[1] Proceed. Med.-Chir. Soc., vol. i.

ventricle is very certainly somewhat hypertrophous as well as soft. Percussion discloses nothing, unless, as is often the fact, the flaccid organ be dilated also. The first sound is short, flapping, weak, toneless, almost to extinction; the second weak and thin; there is no murmur.

The pulse, irregular in force, may be excessively so in rhythm—no connection of time being traceable between it and the systoles of the heart. Long since M. Louis showed that the sudden supervention of these characters in the pulse of patients, laboring under typhoid (Peyerian) fever, was distinctly significant of an acute failure of consistence in the organ.[1] But though, when acutely produced under circumstances of pyrexia, such as these, softening is invariably attended with increased frequency of pulse, where the change is slowly effected by a chronic process, the pulse, though more or less feeble, fluttering, and small, is not of necessity very frequent—the frequency may oscillate a little on either side of the par of health. There occur every now and then cases in which the beat ranges very materially below the average—an infrequency which, though occasionally merely a deception, in consequence of weak systoles not impressing the distant vessels, is not always to be thus explained.

642. *Symptoms.*—There are few symptoms that can be distinguished from those of dilatation. General languor, weariness, incapacity for exertion of any kind, failure of strength, sallowness and lividity of the face, feeble palpitation, sighing respiration, protracted fits of dyspnœa, and painful sensations about the heart, occasionally actual angina, are common to the two affections. Dropsical effusions probably do not occur, unless there be dilatation in addition to alteration of the blood: but the fact is, softening of any duration is rarely unaccompanied with some increase, general or local, of capacity of the cavities. Softening is always a serious condition: it aggravates the ill effects of any other heart-affection it may coexist with, and has of itself proved suddenly fatal, with or without symptoms of angina.

643. *Diagnosis.*—The feebleness of impulse, without increased area of dulness, and the feeble irregular pulse, without the signs of valvular, especially of mitral, disease, are the points on which the diagnosis of softening turns; but these characters will not distinguish simple from fatty softening—and scarcely from dilatation.

644. *Treatment.*—In the management of the disease, an attempt must be made to improve the nutrition of the heart by raising the powers of the system generally, and improving the blood. Animal food, port wine in moderation, pure bracing air, regular but very gentle exercise, and attention to the state of the skin, are the hygienic means indicated. To tonics, quinine, iron, and mineral

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[1] Fièvre Typhoïde, t. ii. p. 274, ed. ii., 1829. The observations of Dr. Stokes on the state of the heart in "continued fever" are also full of interest.

acids, we must mainly trust among medicinal agents. Astringents, provided any ill influence on the bowels can be obviated, might be advisable—among the number, gallic and tannic acids.

Quietude, mental and bodily, is indispensable; too much exercise is infinitely worse than none at all; indeed, the patient's instinct, as well as his feebleness, leads him to disobey the instructions, sometimes given in such cases, for forced pedestrian exercise. Fits of passion should be studiously avoided. Direct sedatives of the heart cannot be given without extreme danger; and inhalation of æther or chloroform is scarcely permissible.

B.—INDURATION.

645. I have never met with a satisfactory example of increased hardness of the muscular structure of the heart, unattended with other textural change. Even in cases of hypertrophy, with hardness, this character belongs, we have seen, not to the actual sarcous structure in itself, but arises from the close packing of fibres of ordinary consistence [588]. I do not precisely understand to what condition Laennec's description of induration of the heart refers. Otto, speaking of "general inflammatory hardening," clearly designates infiltration with induration-matter.

646. The essential causes, and they rarely come into play, of induration, are contractile inspissation of solidified lymph, and calcification. Even these conditions are practically known in the local form only.

VI.—Adventitious Products.

Class I.—Non-Plastic Products or Precipitates.

A.—CALCIFICATION.

647. Precipitation of calcareous salts may occur in any one of the tissues of the heart, as well as in the actual substance of fibrinous coagula, of atheroma, of tubercle, and of induration-matter seated in or upon those tissues.

Including all these possible seats of precipitation, specimens of all varieties of form in the saline materials may be found—granular, nodular, lamellar, and others. In the lamellar form, whether implicating the valves or the pericardium, it is that the imitation of ossification is closest, but neither histologically nor chemically is true bone ever formed.

648. The effects of calcification vary of course with its seat. In the valves it produces obstruction and regurgitation; in the cavities its effects coincide with those of polypoid blood-concretions; in the pericardium, and still more in the heart's substance, calcification probably obstructs the heart's movements; in the coronary arteries, especially if carried to any extent, it must interfere with the nutrition of the organ, and bears a certain, though as yet ill-defined, relationship to the phenomena of true angina.

649. I do not know of any sure means of proving that calcification exists in any one of the positions enumerated. The existence of adventitious substance is in many of them of course easily demonstrable—but to say that that substance is or is not in part or wholly calcareous exceeds our powers. Certainly we possess no means of distinguishing calcification of the endocardium from other morbid changes, interfering originally with the play of the valves. Again, it is *à priori* conceivable that calcified induration-matter in the pericardium should give an osteal quality to the percussion-note over the heart, and impress something of a similar character on the systolic sound—conditions which, with a previous history of pericarditis, might give an inkling of the existence of the state in question. But in actual practice I doubt much whether such signs will ever be found. In a recent remarkable case,[1] where pericarditis was known to have previously existed, the only unusual condition noted was a slight, single, short, commonly systolic rubbing sound; this was referred to the attrition of the material of pericardial adhesion. But certainly I was unprepared for that which the *post mortem* examination revealed as its cause, namely, a ring of pseudo-osseous calcification in old pericardial exudation-matter, an inch broad and about two lines thick, surrounding the heart at the base.

B.—FATTY PRECIPITATION.

650. Precipitation of fat occurs in connection with the heart under two conditions essentially distinct in nature. In the one fat forms in excess in the areolar or connective tissue of different parts of the organ—local obesity results; in the other, precipitation primarily occurs within the sarcolemma of the heart's fibre. The former condition may be distinguished as fatty infiltration, the latter as fatty metamorphosis.

I.—FATTY INFILTRATION.

Infiltration of fat occurs in the connective tissue in three different positions—under the pericardium, amid the muscular fibres, and under the endocardium.

SUB-PERICARDIAL AND INTER-MUSCULAR FATTY INFILTRATION.

651. Sub-pericardial fat chiefly gathers about the base and the right side of the organ, and, no matter on which side, on the ventricle rather than the auricle. When abundant, it encroaches on, and to a greater or less depth renders the muscular texture beneath soft and atrophous. But this influence on the nutrition of the fibres is produced by simple pressure, not by intrinsic deterioration of the sarcous elements themselves.

In this condition there is not a mere precipitation of oil from the blood, but an attempt, more or less complete, at the production of adipose pseudo-tissue.

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[1] Jones, U. C. H., Males, vol. xvii. p. 315.

652. *Symptoms.*—Laennec maintained that this affection "must exist in a very great degree before it gives rise to any serious complaint;" and he was in all likelihood right. True, cases have been recorded in some number, where the gravest effects have been assigned to such fatty disease; but very probably it must either in these instances have led to total destruction of fibre in limited places, or intra-sarcolemmous fatty change must have coexisted. There are no recorded proofs, as far as I know, that rupture of a fatty heart has ever taken place, unless where the latter condition was present, though the accident very conceivably may have occurred independently of this.

Still fatty accumulation under the pericardium produces minor annoyances and some physical signs. Thus, I repeatedly substantiated the existence of the following symptoms and signs in the only person, a male, aged sixty-four, I happen to have watched professionally during life and opened after death, whose heart was at once loaded with sub-pericardial fat, and positively free from serious softenings or notable amount of intra-sarcolemmous oil. Sensation of oppression, or even pain, about the præcordial region; syncopal feelings on exertion; inability to walk quickly on level ground and to get up hill, except with great and painful effort; inclination to coldness in the extremities; feeble (but, as far as I positively observed, regular) pulse, of about medium frequency; sluggish action of the liver and bowels; occasional giddiness; and feeble cardiac impulse, with a too extensive dulness under percussion, the sounds, especially the first, being weak and toneless. There was in this instance a considerable quantity of fat in the lower part of the mediastinum, which may have contributed to weaken the heart's shock against the side. Dr. Hope speaks of the pulse being irregular in such cases; but in the only instance where he made a post-mortem examination, the tissue of the organ was softened as well as loaded with fat. The case above referred to proves that irregularity of pulse is, at the least, not a necessary attendant on the disease.

SUB-ENDOCARDIAL FATTY INFILTRATION.

653. Minute pellets of fat are sometimes seen under the endocardium; in the present state of knowledge they are devoid of clinical interest. I have also frequently noticed, microscopically, fat, granular and oily, between the laminæ of the aortic valves, and also in the substance of fibrinous vegetations on the free surface of various valves; but the clinical import, if any, of these conditions, has yet to be worked out.

II.—FATTY OR OILY METAMORPHOSIS.

654. Fatty metamorphosis of the muscular fibre of the heart, oil being precipitated in lieu of wasted sarcous elements within the

sarcolemma, is an affection of much deeper interest and importance than that we have just briefly discussed.

655. The knowledge that voluntary muscle may be to all appearance converted into fat, is at least as old as the days of Aristotle. Corvisart, however, was the first to teach that the heart might undergo a similar change, and, without having actually seen the disease, to insist on the importance of distinguishing fatty "transformation" from fatty accumulation amid the fibres.[1] Laennec, in turn, not only actually saw, but so clearly described the distinction between this form of destruction and mere fatty infiltration, with muscular wasting produced by pressure, that to his narrative of the naked-eye appearances modern observation adds but little.

656. *Anatomical characters.*—The metamorphosed tissue is more or less deficient in consistence, tearing in extreme cases with the greatest ease, varies in tint from a delicate faded-leaf hue to a pale dirty brown, mottled with darker spots, and rarely feels greasy, though readily yielding oil under pressure. The organ, in most instances natural in size, may be hypertrophous, or, as is much more common, dilated, locally infiltrated with blood, aneurismal or actually ruptured. The coronary arteries may be atheromatous, calcified and obstructed, or healthy; nor does there exist any necessary relationship between morbid conditions of these vessels and the muscular waste. The aortic or mitral valves, or both, are sometimes diseased; but there does not seem, from what has fallen under my notice, to be any necessary or even close connection between the two states; neither does calcification of the aorta appear to be more frequent than in persons of the same age cut off without fatty heart.

Both ventricles are most commonly affected, the left alone more frequently than the right alone. The layers of substance immediately beneath the endocardium and pericardium commonly, but by no means always, suffer earlier than the intervening strata. The diseased change may be very distinctly limited to particular strata or islets of the ventricular substance for a variable length of time; eventually the entire thickness of the walls, papillary muscles, and columnæ carneæ become involved. The auricles seem very little prone to this morbid change: Dr. Ormerod questions its ever occurring there, but I have most certainly observed it in the right auricle.

The microscopical characters of the disease are closely the same in the heart as in voluntary muscles. But its very earliest evidences are perhaps less rapidly caught, because, as Dr. Ormerod observes —an observation in which I concur—the transverse striæ are in the normal state less distinctly marked in the cardiac than in the voluntary fibre: this, at least, I hold to be true of the adult heart. Be this as it will, such striated arrangement, as normally exists, loses its clearness of definition—the actual sarcous substance gives place to fat granules and to oil globules, which granules and globules, at first disposed in more or less evenly transverse rows, eventually lose all regularity of arrangement, and seem to take the place molecule by molecule of that substance. The nuclei of the fibres earliest disappear. The addition of ether, which dissolves the oil (an addition imperatively required for the sure distinction of the granules and globules from saline particles and from starch), will disclose elements of fibre still remaining intact in spots, where heaped-up granules had rendered them invisible. The diseased fibres are not notably, if at all, less in diameter than healthy ones.

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[1] Maladies du Cœur, 3d edition, 1818, p. 180.

Oil is also found outside the sarcolemma, probably in part originally precipitated there, in part reaching that situation from within the sarcolemma either through ruptures from over-distension or through perforations resulting from atrophous absorption. As a rule, it must be remembered, the sarcolemma continues discernible, and hence the outline of the fibre is preserved; but where the destructive process has reached the ultimate point possible, this membrane itself disappears, whether by mere atrophous absorption or by fat-conversion is not clear.

Fatty metamorphosis of voluntary muscle is accompanied with accumulation of oil within the neurilemma of its supplying nerves; whether the nervous structure of the heart undergoes a similar change is imperfectly known.

657. Little advance has been made in the local chemistry of the disease. Dr. H. Weber comes to the startling conclusion that there is less fat chemically discoverable in heart-substance to all appearance fattily disorganized, than in healthy texture, in the ratio of 1.9 : 3. Despite my personal knowledge of Dr. Weber's great accuracy, as an observer, I cannot help suspecting error must somehow or other have crept into his inquiry.

658. *Coexistences.*—Fatty metamorphosis has been found in hearts of which either the external or internal membrane, or both, had been the subject of former inflammation. There seems fair reason to suppose the adjacent inflammation process may in such cases have really promoted the mal-nutrition of the muscular substance. It is unlikely the nutrient processes could maintain their par of health, while textures so closely connected were passing through exudative and other changes. And more than this, as we have already seen [466], fatty change does sometimes appear to occur as a dependence on acute pericarditis—and this under circumstances apparently proving the non-existence of any prior chronic affection of the heart.

But beyond this I know of no local or diathetic state that experience specially connects with fatty disorganization of the heart. All varieties of acute and chronic pulmonary inflammation, empyema, and emphysema of the lungs may be found in cases of the disease—there is no relationship of cause in one direction or the other. I have seen the disease in association with hepatic cirrhosis, with faded-leaf fatty liver, with true lardaceous or cholesteric liver, and with gall-stones—but it is almost needless to add the heart may be sound in all three. The frequency of the disease in phthisis remains to be calculated; but it is certainly not great: my own experience does not furnish me with a single example of advancing phthisis coexistent with fatty metamorphosis of the heart carried to an extent to produce notable symptoms. Chronic colorless softening of the brain, as also of the spinal cord—acknowledged effects of mal-nutrition—are sometimes found in association with it.

Again, persons affected with Bright's disease, scurvy, purpura, gout, chronic rheumatism (without inflammation of the cardiac membranes), and the hemorrhagic diathesis, occasionally suffer from the affection; but, with the exception of gout, there is not a single one of these diathetic diseases that has attracted my observation by frequent coexistence. True, some of them are rarely witnessed in London practice. Nor, as far as my reading goes, has that singular diathetic state of muscle, Cruveilhier's "progressive atrophy," any tendency to attack the heart.

On the whole there seems to be no diathesis which positively promotes or antagonizes this structural change, nor, *per contra*, does fatty degeneration in the heart clearly encourage, or exclude, the development of any other form of disease: it may prove, death occurring at an advanced age, the sole textural defect of practical significance in the body. Although sometimes combined with fatty infiltration, there is no necessary connection between the two things; rather the reverse indeed. And, further, general corpulence with local obesity of the heart may exist in the highest degree unaccompanied by true fatty metamorphosis; while in thin, almost emaciated frames, the specific fatty deterioration of fibre has occasionally been found.

659. *Intimate nature and mechanism.*—It would be foreign to the purposes of a purely practical work to enter upon a full discussion of the mechanism of fatty degeneration—as exposed variously by Paget, Ormerod, Quain, Lehmann, Valentin, and Handfield Jones. But it may be as well to state my belief that the recognized laws of vital processes, as well as attainable positive evidence, are repugnant to the admission of the direct metamorphosis of proteinaceous into fatty matters in the midst of a tissue living and active—as of so much dead material into adipocere. They who write concerning saponification of the heart as a conversion easy of accomplishment, should study the observations of Orfila,[1] whose exhumations prove that even in the dead tissues the process is one requiring very considerable time, and a number of favoring conditions. Without venturing to deny the possibility of occasional direct chemical conversion of effete devitalized myoline into oil, I believe such change

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[1] Exhumations Juridiques.

to be purely accidental, and that Nature's ordinary mode of procedure is this:—first, atrophy of the sarcous elements occurs, and secondly, oil is molecularly laid down in the place of the absorbed elements. The fat is the mere index, in no wise the essence, of the disease.

Hence the true problem becomes the determination of the causes of the primary failure of nutrition of the sarcous structure: and of this we are utterly ignorant. Analogy helps us little. Thus the fatty pseudo-metamorphosis of muscles adjoining non-reduced dislocations and ankyloses, and that occurring in paralyzed limbs, and in bedridden people, is explicable by the waste consequent on inaction: but it is difficult to admit even the minor influence of any such cause in the case of the heart. Nor, again, does the fatty pseudo-metamorphosis of uterine tissue, which follows parturition, lend aid in the explanation. Now, direct evidence, as far as it goes, deposes against the merely local mechanism of the cardiac decay; there is no necessary relationship between a diseased state of the minute vessels or nerves of the heart and its failing nutrition. On the other hand, we are unacquainted with any form of blood-disease or nervous dyscrasia even seemingly essential to the existence of the disease. Dr. Smith, of Dublin, it is true, in some cases of the kind, found oil in the blood; but can we, with Dr. Stokes, accept the fact as an "apparent proof" that fatty heart may be produced by the assimilation of ready-formed oil in the blood? I am disposed to think not: the two conditions were probably mere coincidences. Simon's piarhæmia may exist without fatty heart, and fatty heart without that diseased state of the blood. And admitting the reality of some, as yet undiscovered, preliminary morbid state of the blood, it would remain to be ascertained why that particular form of pravity of the fluid should lead to the impairment of the nutrition of the heart rather than of other textures.

Clinically, fatty pseudo-metamorphosis is closely allied to fatty infiltration of the connective tissue, with pressure-atrophy of the intervening muscular fibre; pathologically, there is this difference, that in the latter, fat-formation is primary, sarcous atrophy its effect; in the former, the myoline primarily decays, and oil is laid down in its place. This at least I believe to be the general truth; but doubtless, as ably argued by Dr. Handfield Jones,[1] in some cases this distinction does not hold—the formation of extra-sarcolemmous fat being itself the consequence of initiative atrophy of fibre.

660. *Predisposing causes.*—Although fatty decay of the heart has occasionally been seen in young persons, the disease is essentially an apanage of middle and advanced life. Males exhibit it, both in its slighter and graver degrees, with considerably more frequency than females. It occurs in all ranks of society, though, to a certain

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[1] Fatty Degeneration, Brit. and For. Med.-Chir. Rev., April, 1853, p. 334.

but undetermined extent, more in the upper and middle classes than among those who earn their daily bread by manual toil. The influence of special kinds of trade or occupation, if any, has yet to be discovered.

Aristotle ascribed conversion of voluntary muscle into fat to excess of nourishment; modern observers add the abuse of alcoholic fluids, lazy habits, and luxurious living generally, as at least favoring the metamorphosis. But I doubt very much from my own observation whether these conditions can be fairly held to play any very notable part in the instance of the heart. True, I have seen the disease in persons of sedentary, luxurious life; but I have met it also in professed pedestrian tourists, and in persons habitually and actively employed. I have seen it in men whose rule for years had been to consume at least their daily bottle of wine, in gross beer drinkers, and in spirit drinkers; but I have observed it also in persons whose life had been one, not only of soberness, but almost of abstinence.

Yet more, there is no doubt that fatty disintegration is sometimes seen to a very considerable amount in the hearts of men, who at, and long prior to, the time of death, have systematically lived after the most approved hygienic models. All this forces on us the conviction that in this, as in most chronic disorganizing diseases, the real predisposing cause is a certain *constitutional aptitude*, formed out of intrinsic peculiarities, congenital or acquired, of structure and function, that lie beyond the pale of our existing powers of scrutiny.

661. *Clinical conditions.*—The clinical aspects of the disease vary greatly with the superficial extent and the degree of the atrophous change. That fatty metamorphosis may be found, and to no insignificant amount, where neither subjective nor objective cardiac symptoms had awakened attention during life, is indubitable. And it is equally certain that hearts have been carefully examined during life, and pronounced free from disease, which almost immediately afterwards (death arising from other causes) have been found very sensibly fatty. The disease may then to the observer of the present day, at least in its minor and moderate degrees, be *latent*. But the rule is that, if the affection be at all marked, its clinical evidences are more or less precise and positive.

662. *Physical signs.*—The physical signs are those of a heart soft, without notable enlargement. The impulse, always weak, is in extreme cases wholly imperceptible—the precise point of the apex-beat, though ill-defined, can be caught better, both with the eye and finger, than in cases of attenuated dilatation with flaccid fibre. The existing indistinctness of the apex-beat is probably owing to the want of power of the fibres, naturally giving the spiral movement to the heart in systole: the whole organ is simply, and as it were lazily, projected forwards.

The cardiac percussion-dulness, both superficial and deep, remains unchanged in area, unless there be alteration of bulk from some independent cause.

Auscultation discovers, as a general rule, a weak, but by no means always toneless, short and relatively high-pitched, first sound; a long first silence, and a feeble, but relatively distinct and accentuated, second sound. If the fatty disorganization be, as it often is, in great excess in the left ventricle, the first sound may be of notably fuller tone at the right than the left apex, and the second sound louder and more strongly accentuated at the second left, than at the second right, cartilage: the relative state of preservation of the muscular structure of the two ventricles explains these facts. I have never met with absolute deficiency of either sound over the entire cardiac region; but it is not uncommon (quite independently of the presence of any apparent syncopal condition) to find the first sound at the left apex almost null from weakness. Possibly a dynamic mitral regurgitant murmur may sometimes occur; but I do not know this from observation; and, in the present state of knowledge, if murmur exist, its explanation must be looked for in valvular disease or alteration of the blood. At least, I formerly thought thus; but experience obliges me to modify this statement of the former editions. I have for some time had under observation a case in which the whole symptomatology points to the existence of moderate fatty disintegration, and in which sometimes at the periods of syncopal and pseudo-apoplectic seizures, sometimes not at those periods, a most distinct though feeble systolic murmur becomes temporarily audible at the left apex.

663. *Symptoms.*—Nothing can be more variable than the mode of grouping, or than the intensity of individual symptoms. Between the extremes of latency, on the one hand, and conditions of cardiac, encephalic, and pulmonary function so perverted that the continuation of life seems an impossibility, on the other, all conceivable shades of degree are observed. We must assume in our general outline of the patient's state, that the characters of the disease are, if not highly marked, at least well and distinctively defined.

The patient whose heart is notably fatty, though not of necessity in a low state of general health, is unable to undertake any sustained labor, being exhausted on the first attempt almost, and forced often, when making an effort to walk, to stop at every few paces from cardiac uneasiness or dyspnœa, or both. In temper he is dejected and peevish—emotional states coming either directly of the disease and its perverted state of circulation, or indirectly of the incapacity for occupation it entails. His tissues generally are soft and flabby; his muscular power almost null; the skin of the face pale and sallow, or more or less livid; the feet and ankles disposed to slight, but rarely, if ever, the seat of grave, œdema [617]; the appetite feeble, the digestive forces languid, the bowels confined, the liver larger than natural, and readily increased in size during paroxysms of dyspnœa.

The state of the respiration varies widely in different cases, and at different times in the same case. The patient, though easily put out of breath, as a rule, and subject to fits of dyspnœa of very irregular rhythm and sometimes orthopnœal, yet not constantly asthmatical, may, on the contrary, be free, even under the pressure of great cardiac distress, from subjective or objective difficulty of breathing. So, too, may similar freedom of respiration hold on the eve and during the occurrence of the syncopal and apoplectic seizures incident to the disease. A tendency to sigh, without emotional influence or mere habit to explain it, is in some cases connected with fatty disintegration. Again, in certain instances the breathing exhibits very peculiar characters, in regard of rhythm and intensity, first described, as far as I know, by Dr. Cheyne, in 1816. A series of inspirations takes place of gradually increasing force, at a rate of speed, slightly accelerating as they proceed, until a certain maximum is reached. An ascending series being thus completed, a descending follows of force gradually decreasing, until respiratory sound can scarcely be caught through the stethoscope. This immediately precedes a period of apnœa—neither chest motion nor breathing-sound being perceptible. So complete, indeed, is the inaction of the lungs, that were not the pulse felt, the patient might be supposed dead, until a low inspiration marks the commencement of a new ascending and descending series of breathing acts. Beyond the changes in intensity and rhythm there is nothing unusual in the physical signs—no sibilus or other indication of spasm of the bronchia. Neither is there, as far as I have seen, any evidence of subjective respiratory distress, or, indeed, of suffering of any kind during the suspension of breathing. But freedom from suffering does not, however, always appear to hold during the entire paroxysm. Dr. Stokes speaks of "every muscle of inspiration being thrown into the most violent action, accompanied often by a loud moan from the patient."

In Dr. Cheyne's case, each revolution in the state of breathing occupied about a minute; the apnœa a quarter of a minute; the number of respirations in a minute equalling about thirty. In a case observed by myself some while since,[1] the apnœa lasted thirty seconds on each occasion—the recurrence of the fits of perverted rhythm being irregular. The phenomenon may continue for months in a minor degree, and may occur during sleep: at least the testimony of the patient's wife, in the case just referred to, was very positive on these points.

This singular condition of breathing is of extreme rarity. What is its mechanism? An observation of Professor Seaton Reid, to be by-and-by noted, shows that it has no specific connection with fatty disintegration *per se:* as far as the heart's substance is concerned, weakness is probably the only necessary element. A gradual weakening of the action of the right ventricle suggests itself in explanation—the blood stagnating in the lungs until the instinctive craving for oxygen again sets the apparatus in feeble motion. But the pulse at the wrist holds on unchanged during the paroxysm; why should the right ventricle fail in action, while the left thus proves itself to be vigorous? Besides, in the case above referred to, jugular pulsation continued in the neck, and the sounds at the pulmonary cartilage maintained their fulness, during the apnœa. Neither are there any conditions of brain present (unless such pre-existed), during the entire fit of perverted rhythm, pointing to stagnation within the cranium. It probably lies beyond the powers of existing physiology to explain satisfactorily the mechanism of this apnœa; but, guided by the facts just referred to, I cannot avoid inferring that the proximate cause lies in a failure of the special nervous excitant of the respiratory act—in anæsthesia either of the vagus or of the medulla oblongata itself. The perversion of rhythm, though differing in character, is probably allied in mechanism to the state I have elsewhere described as "nervous apnœa."[1]

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[1] In conjunction with Dr. King and Messrs. Elliott and McMeikan of Stratford.

I have known cough a troublesome symptom, unattended with signs of bronchitis or any other pulmonary affection.

Seeing that pulmonary apoplexy is an occasional effect of flabby or of hypertrophous dilatation of the left ventricle (conditions tending to stagnation within the heart), it would follow that the weakness of fibre in fatty metamorphosis should conduce to blood-infiltration in the lung. Still, as matter of fact, I have not seen this, unless where dilatation coexisted.

The pulse may be perfectly regular in force and rhythm, and of medium frequency, as the habitual state, even where strong evidence exists of advanced fatty change—nay, in such cases, even when the pulse becomes paroxysmally frequent, with anginal pain, it may maintain its regularity. But this is the exception. Commonly the pulse is irregular in force and rhythm, either constantly or from time to time under the influence of excitement, flatulence, indigestion, or effort. On such occasions it may become exceedingly frequent; I have known it uncountable—in the main from frequency; in part, however, from irregularity. The pulse may, when frequent and irregular, have also a tingling character, according to Dr. Stokes; but in the only instance he reports there was marked mitral coarctation. A third, and more noteworthy and special condition of the pulse is infrequency—sometimes carried to a very remarkable extreme, the beat falling, for instance, to twenty-six per minute.[2] Such infrequency is in some cases referable to extra-weakness of occasional systoles; but, as originally noticed by Dr. Adams, the ventricular systoles themselves are occasionally much less frequent than natural; and, in point of fact, defect in the cardiac action itself is the really important condition. The infrequent beats may individually be either slow and lagging, or quick and abrupt.

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[1] Diseases of the Lungs, 3d Am. edition, p. 252.

[2] J. B., private cases, May, 1854.

The mechanism of infrequent action has already been discussed, and the mystery attached to it admitted [342]. But it is yet more difficult to fix what are the conditions in different cases specially productive of the three main varieties of pulse just described. The greatest irregularity may exist without mitral disease, but not, I am inclined to think, without dilatation.

Occasional fits of palpitation, attended with choking sensation, cardiac uneasiness, pain or actual angina, occur; and the patient, readily becoming faint on the least exertion, falls into a state of actual syncope from time to time.

Little or nothing is known positively concerning the state of the blood. I have failed to find the venous hum of anæmia, in cases where the tissues were markedly pallid—but the patients were aged. The piarhæmia, noticed by Dr. Smith, was clearly exceptional and accidental.

It is more than doubtful that the urine possesses any distinctive, or indeed habitually morbid, character: I can answer in the negative for fat, albumen,[1] sugar, biliphæin, excess of urea, casts of the tubules, and spermatozoa; oxalates, uric acid, and amorphous and triple phosphate, occasionally present, are of no significance, except that they may point to the coexistence of an independent diathesis. As to the quantity of water habitually eliminated by this channel, without being able to furnish as precise information as I could wish, I can distinctly affirm that it does not oscillate in either direction notably out of proportion with the fluids consumed. This clinical truth seems somewhat at variance with the theory of eccentric pressure on the renal capillary system already referred to [593, *h*].

Sexual inclination and power may both fail—the latter more than the former. In one of the best marked cases of the disease, clinically speaking, I ever saw in a male under the age of forty-five, it was this failure, and not the cardiac symptoms, that led to my being consulted. Here, too, the genital organs were sound, and there existed neither spermatorrhœa nor spinal disease. Was the failure a mere coincidence, or really an effect of the languishing activity of the heart? On the other hand, I have seen many cases in which, as far as I could ascertain, there was no lack of erotic propensity or copulative vigor.

The functions of the encephalon very habitually suffer more or less grave impairment. Little doubt can be entertained that in many cases formerly set down as examples of pure dynamic disturbance of the brain, the groundwork of this lay in perverted circulation dependent on fatty metamorphosis of the heart.

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[1] Slight albuminuria occurs where dilatation coexists with fatty change; but I have not known the latter, unaided, produce it.

As a general description it may be said that vertigo, failure of sight, dull aching and variously anomalous, indescribable sensations in the head, feebleness of intellect, and especially of memory, and somnolence, become common, with the advance of the disease, either as almost habitual states, or as paroxysmal occurrences.

The failure of memory is particularly striking—like that attending chronic softening, it bears on recent rather than long-past events. The psychical state, immediately preceding the syncopal attacks appertaining to the disease, is sometimes very curious. Thus a very intelligent patient, who has had a considerable number of such attacks, says: "For a minute or so before unconsciousness takes place, my memory of things just passed goes: I remember invariably, or rather my fancy conjures up, one of two or three different scenes that I have been witness of; there is no delusion, mental or optical, for I know they are not before me; but I think by shutting my eyes, I could realize them."

But these forms of disturbance may proceed much further. The patient, suddenly losing consciousness, may exhibit many of the appearances of an apoplectic seizure—sudden fall to the ground, complete insensibility, with breathing either stertorous or of the peculiar form previously described, while the pulse becomes, even if previously an infrequent one, still more infrequent. The attacks are generally brief, easily recovered from, are very rarely followed by paralysis—and in their immediate mechanism appear to me, with Dr. Stokes, rather to depend on deficient arterial supply and venous stasis, than on excess of blood transmitted to the brain. Dr. Stokes refers to a case in which the patient, warned by certain premonitory symptoms, was enabled to save himself from attacks by "hanging his head, so that it rested on the floor." There seems, indeed, a close alliance between these apoplectiform attacks and syncope; and a patient who has long been the subject of syncopal attacks, may lose these and have the apoplectiform variety instead. But, more than this, the seizure may be of a mixed apoplectic and convulsive character—clonic spasms of certain parts coinciding or alternating with tonic spasms of others. Or the syncopal and apoplectic elements may be wholly deficient, the seizure being epileptiform in attributes—marked by sudden fixed gaze, death-like pallor of face, clonic spasms of the muscles of the head and neck, subsequently of the limbs, biting of the tongue, dilatation of the pupils, and on recovery total oblivion, or rather ignorance, of all that has passed during the paroxysm. I do not happen to have seen foaming at mouth, nor to have heard the epileptic cry, in attacks of this particular kind; but I have had the affirmative testimony of friends of patients as to the occasional existence of both.

Again, I have known a patient of this class, while engaged in ordinary occupation, sink on a chair, without actually falling, bend forwards as if to relieve suffering of some kind, become at the same time of deathly pallor, and rapidly lose heat at the head and extremities, and utter no cry, while the pulse remains perceptible, no muscular twitch even occurs, and the pupils do not dilate. The consciousness goes completely in the objective, but apparently not wholly in the subjective sense; waking up in a state of bewilderment, after a lapse of some three or four minutes, the patient speaks of pain at the lower sternal region, of which pain *all memory disappears after a few minutes more.* Strange combination of cerebral and cardiac disturbances—and which in more aspects than one seems to represent the state artificially induced by chloroform.

Now, the usual and perfect recovery after seizure in all these varieties would of itself suffice to show that they are unconnected with structural change in the brain. Neither is the converse form of proof wanting, for death has occasionally taken place after a repetition of them, and the cerebral substance been found free from any one of the admitted conditions of textural disease. Atheromatous change in the capillaries is doubtless sometimes present, and may probably aid in promoting the blood-stagnation, on which the seizures in some cases depend. On the other hand, when, after apoplectiform seizure in a case of fatty heart, imperfect hemiplegia, quasi-idiocy, and mutism (as I have known), hold on for months, the reality of organic change cannot be questioned.

The epileptiform character of the attacks would suffice in the opinion of some persons to prove implication of the spinal cord. In addition to this evidence I may state that in two cases, where abundant evidence of fatty metamorphosis existed, I observed formication, slight numbness, and tendency to subjective sense of cold in the lower extremities, the patients being free from other ordinary symptoms of disease of the cord, and certainly free from intra-pelvic affections, enlarged prostate, stricture of the urethra, renal disease, and spermatorrhœa. Were not these symptoms of implication of the medulla analogous in mechanism to the quasi-apoplectic seizures?

664. *Course.*—As a general truth, fatty metamorphosis is a chronic process—that is, though the immediate change, molecularly considered, may be, and probably is, rapidly effected, the extension of the change to any notable amount of structure, and its capability of producing appreciable symptoms, are the work commonly of months or of years. Nevertheless the fact, that individuals have occasionally been met with, noted for muscular prowess and feats of endurance almost to the day of their death, whose hearts notwithstanding have proved the seat of fatty conversion on a most extensive scale, seems to depose in favor of rapidly acute course as at least an occasional occurrence. And, as already stated, under the influence of adjacent inflammation, it seems tolerably certain fatty disintegration of the fibre may be acutely effected. On the other hand, it is conceivable that in the exceptional individuals just referred to (specially organized they must in any view be considered to have been), the supply of *vis nervosa* to the heart may be so great, as to render the

contraction of a small mass of their heart-fibres as effective as the sum-total of those of ordinary persons. See, in the way of analogical illustration, the muscular work achieved by a feeble anæmic woman in hysterical paroxysm. But this important point in the history of fatty disease requires *ex professo* clinical study.

665. *Manner of death.*—Patients, whose hearts have undergone fatty metamorphosis, are often possessed with the idea that they shall expire suddenly. For this idea there is commonly a very obvious foundation, in the fact that they have been more or less subject to various cardiac paræsthesiæ, or to actual syncopal attacks; but occasionally no such subjective hint has been, at least appreciably, given to the sufferer. In both classes of case the idea occasionally proves a prophetic one.

Instantaneous death may be of purely dynamic mechanism—sudden syncope occurring from failure of the heart's intrinsic irritability, or from failure of motor innervation, or perhaps from excess of inhibitory innervation. Or such death may be mechanically caused by rupture and copious extravasation into the pericardial sac. Or, in cases of partial or minute rupture, death is at once statically and dynamically brought about. Rapid (but never actually instantaneous) death may follow apoplectiform and epileptiform seizures.

In many instances the fatal event is slowly brought about by asthenia.

On the other hand, fatty disorganization of the heart is by no means necessarily fatal. I have known extensive destruction of the kind exist, where death had occurred from unconnected chronic disease of other organs; and the proofs that a moderate amount of the change may be comparatively innocuous are of daily occurrence. Nevertheless, could we positively diagnosticate the disease, no matter how slight its extent, its existence should never be lost sight of, either in regard of prognosis or therapeutics.

666. *Diagnosis.*—If weak cardiac action, a feeble toneless first sound, with a relatively well defined and accentuated second sound, absence of murmur, and percussion-dulness of normal area, coexist with frequent and irregular or infrequent and regular pulse, respiration of ascending and descending rhythm, and the peculiar cerebral attacks described, there can probably be little doubt of the existence of this form of atrophy of the heart. But the value of this proposition is less than it seems; for, even in cases anatomically well marked, the pulse may be perfectly natural; apoplectiform attacks are very rare; and the perverted rhythm of respiration equally so.

Besides, it seems now established, as I suggested in the edition of 1854 would probably prove to be the fact, that other forms of cardiac disease, without a particle of fatty metamorphosis, may produce the special rhythm of breathing—and it is very probable, also, the peculiar apoplectiform seizures. In fine, the physical signs enumerated, coupled with the deficiency of the dropsies and other systemic effects of dilatation, seem to furnish the most serviceable guides to direct diagnosis. Some help may occasionally be had from the notable perversion of the pulse-respiration ratio, entailed by the relative infrequency of the heart's contractions. Thus a pulse of forty may co-exist with a respiration of twenty per minute, or a ratio of 2 : 1.

Mr. Canton shows the arcus senilis to be the result of fatty atrophy of the cornea. But the arcus senilis, like fatty heart in the generality of cases, is an attribute of age: its discovery will do no more in confirmation of an otherwise doubtful diagnosis of fatty heart, than would ascertaining that the patient has passed his fiftieth year. If indeed the corneal change were found in a youthful person, it might possibly have some weight: still, that fatty heart may exist without change in the cornea is certain—and the converse holds equally good. The most remarkable specimen of fatty opacity of the cornea I ever saw in a person under forty-five years of age, so remarkable that the grave disfigurement attracted the attention of non-professional persons, occurred in a male cut off by chronic alcoholism, cirrhosis of the liver and ascites, who never exhibited the least clinical evidence of fatty disease in the heart.[1]

The direct diagnosis of the disease is consequently in an unsatisfactory state enough; nor is the clinical difficulty of the case always removed by contrast of the conditions of this affection with those of others.

Thus, positively distinctive signs of simple flaccid softness and fattiness of texture are in very truth wanting; and the chief safeguard against error lies in the extreme rarity of genuine cases of pure softening.

Nor do I know of any means whereby fatty pseudo-metamorphosis may with surety be distinguished from mere infiltration. The researches of M. Bizot into the relationship of subcutaneous fat to fat-accumulations about the heart furnish no clinically available help.

The natural areæ of the cardiac dulnesses, and the absence of systemic congestion and dropsy will distinguish fatty disorganization from dilatation; if there be marked jugular distension, or *à fortiori* jugular pulsation, the case is not one of mere fatty disease.

From all forms of valvular disease the present affection differs in the absence of persistent organic intra-cardiac murmur. But, as we have already seen, there is fair motive for believing that mitral regurgitant murmur of dynamic mechanism may temporarily occur. Basic systolic murmur of blood-origin is very rare; whatever be the morbid state of the fluid in these patients, it does not promote the occurrence of murmur.

From chronic pericardial effusion fatty disease will differ by the normal area and shape of cardiac dulness, by the absence of præcordial arching, and by the fact that the heart-sounds are at least as well marked at the cardiac region as at the top of the sternum; the history of the case will, further, prevent the possibility of error.

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[1] Mr. H., seen with Dr. Donald Fraser.

Supposing all commemorative story deficient, it may prove exceedingly difficult to distinguish one of the peculiar apoplectiform attacks intercurrent to this disease, at least at the actual moment of seizure, from uræmic coma. The breathing may, however, be genuinely stertorous in the former, while it never is so in the latter variety. In the pallor of face and absence of primary or sequential paralysis the two varieties agree. The condition of the urine, and the absence or presence of anasarca, will be the most available, but not infallible, guides to the diagnosis.

Professor S. Reid suggests that notable slackening of the pulse, when the peculiar respiratory disturbance just described reaches its period of greatest effort or "distress," with increased frequency of the pulse during the period of apnœa, is a combination probably distinctive of fatty disorganization.

667. *Treatment.*—The treatment of this affection is essentially that recommended for simple softening of the heart. It seems natural to enjoin abstinence from fats of all kinds, and of materials amylaceous and saccharine, readily converted into fat; yet there may be little philosophy here after all—if the local fatty state be, as seems strongly probable, a mere evolution of a form of atrophy. On the other hand, as to the propriety of a full diet of proteinaceous food of easily digestible quality there can be no doubt. The vigor of the circulation requires sustainment from the moderate use of wine or spirits; and when, as is often the case, sufferers from the complaint have been habitual free livers, it appears to me gravely unwise (no matter what a tentative chemistry of the disease may suggest concerning the influence of an alcoholized regimen) to lessen their wonted daily amount of stimulus.

It is almost unnecessary at the present day to denounce the idea of letting blood in any form or degree for the disease itself, or for its accidental secondary phenomena.[1] Not only this, but the sufferer from fatty heart should be very cautiously submitted to lowering regimen or depressing medicinal treatment for any intercurrent attack that might, in a previously healthy person, call emphatically for such measures. The inhalation of chloroform is very certainly hazardous, though it is impossible to avoid the conviction that people with fatty hearts have occasionally inhaled with perfect impunity. Chloroform liniments may be used to the præcordial surface, provided due caution be observed; they sometimes relieve cardiac distress very notably.

Of medicinal agents, iron, quinine, and strychnia are the most valuable. From the latter I have, as I believe, seen distinct improvement occur in the force of the heart's contraction. On somewhat of the same principle the gentle appliance of electro-galvanism seems seriously worthy of trial; but the necessity of caution in its use flows from statements already made [407].

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[1] Dr. Cheyne bled in cases of fatty heart. Will it be affirmed that the "type of the disease" has changed since his day?

Moderate exercise on level ground, if not productive of distress, should be daily taken. Pure bracing air is theoretically indicated. But I am unacquainted with any facts proving that forced exercise in mountainous countries can cure, or even stay the onward progress of, the disease we have been considering; the cardiac affections I have known removed by Alpine tours have been simply functional derangements of the heart dependent on gastro-hepatic dyspepsia.

The apoplectiform seizures, intercurrent to the disease, should clearly be met by stimulants internally and counter-irritation to the lower extremities: they ought in fact to be treated much as syncopal attacks. Epileptiform paroxysms are plainly to be managed on ordinary principles.

I cannot close this brief account of fattily diseased heart without mentioning my strong persuasion that I have seen more than one instance in which, through hygienic and medicinal measures such as those just mentioned, not only has the progress of decay been arrested, but real improvement actually effected in the nutrition of the heart. True, I should be puzzled to give positive demonstration that changes so notable had certainly been achieved; such demonstration is obviously unattainable. I found my belief on the facts that in certain cases where the physical signs, the local annoyances, and the general dyscrasia pointed strongly to fatty disorganization, and to no other disease, as the cause of all, those signs have improved, those annoyances have disappeared, and that dyscrasia has given place to a diathetic state of relative tone and plasticity.

C, D.—AMYLOID AND SACCHARINE PRECIPITATES.

668. Amyloid degeneration has been seen in the heart and pericardium.[1] The symptoms, where the heart is concerned, will doubtless be those of ordinary fatty change—but experience is limited.

Sugar has not, so far as I know, been found in the substance of the heart. But Bernard some years since detected diabetic sugar in pericardial serosity; the fluid, alkaline at first, became acid through the decomposition of the sugar.[2]

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[1] Friedreich in B. & F. Med. Chir. Rev., Oct. 1860.

[2] Bernard generalizes this observation by suggesting that the excess of acidity in diabetic people, commonly regarded as an element in the causation of the disease, is really a mere effect of decomposition of already formed sugar.

CLASS II.—BLASTEMAL FORMATIONS.

SUB-CLASS I.—DEPOSITS.

A.—SYPHILITIC DEPOSIT.

669. If the heart, like the lung, ever becomes the seat of syphilitic nodular deposit, the fact remains to be proved. Some years ago I opened an individual cut off by tertiary syphilis, whose heart presented appearances suggesting the possibility of productions similar to subcutaneous gummata being formed therein.

B.—TUBERCLE.

I.—CARDIAC.

670. Tubercle very rarely forms in the heart, and only, as a rule, in very small quantity and in cases of general tuberculization, or as an extension from similar substance deposited under the pericardium. Its signs and symptoms, if it have any peculiar to itself, are unknown.

A case, remarkable for its exceptionality in almost all points, was observed many years ago by Townsend of Dublin. Here, while the lungs of the patient (a male aged sixty-one) contained only a few miliary tubercles, a tuberculous mass of some size existed in the walls of the heart, producing eventually fatal pressure on the pulmonary veins just at their line of union with the left auricle. Dyspnœa was the essential symptom.

II.—ENDOCARDIAL.

671. Minute tuberculous masses in the crude state, and also gray granulations, form under the endocardium in infinitely rare cases of general tuberculization in the child. I know nothing of such deposits acting as the cause of endocarditis, though, on the analogy of the pericardium, it is very probable they may so act.

III.—PERICARDIAL.

672. But tubercle is a notably more common, though still a rare, formation beneath the serous layer of the pericardium, either cardiac or parietal. Occurring in the state of semi-transparent gray granulation, it may remain latent, or excite inflammation of that membrane. The ensuing pericarditis produces the same physical signs and local disturbance as if it were of idiopathic origin. But, probably on account of the tendency to secondary formation of tubercle amid the simple inflammatory exudation-matter,[1] the disease, unless fatal at once, tends, just as peritonitis of the same origin, to lapse into a chronic state, attended with more or less constant local

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[1] Carswell's Drawings, U. C. Museum, A. 533, Fig. 2.

symptoms. This was the view of Laennec; and it is supported by the course of a case that fell under my own notice.

673. In ordinary phthisis, as appears from the records of M. Louis, tuberculous pericarditis is very rare: it is vastly more uncommon than pleuritis or peritonitis, and notably so even than meningitis, of that form. It must not be forgotten that a phthisical patient may be seized with pericarditis, which shall be in no wise distinguishable in its products from that occurring in ordinary rheumatism, and which may prove rapidly fatal in the acute stage.[1]

674. *Diagnosis.*—Probably enough miliary tubercles raising the pericardial surface might by their abrupt collision generate a form of friction-murmur; but were such murmur heard, I know of no means by which its mechanism could be determined. In point of fact, in the existing state of knowledge, its discovery would tend rather to mislead than enlighten the observer—as it would obviously simulate the ordinary sign of acute exudation or congestion of the pericardium.

And even this sign, of deceptive import, will commonly be wanting; tuberculous deposit will of itself give no physical evidence of its presence, and the diagnosis of the disease can only be made through the pericarditis it occasionally entails. But would the tuberculous origin of this pericarditis be announced by any peculiarity in its signs? Very certainly not; still, an attempt at divining its character might be made in the following way. If there were neither rheumatism, nor Bright's disease, nor adjoining pleuro-pneumonia present, and the patient had received no injury to the chest, and was distinctly of strumous constitution, the probabilities would be much in favor of the tuberculous origin of the inflammation. And this, too, although there were no distinct pulmonary signs of advancing phthisis; for, herein resembling tuberculous peritonitis, this form of disease has mainly been observed in cases where the lungs were but slightly affected.

But my recent experience has shown me yet another form of difficulty attending this diagnosis. A patient may have pericarditis of the most obviously scrofulous character, and yet not the slightest friction-murmur exist—the absence of the sign resulting, as shown after death, from the excessive, almost greasy, smoothness of the strumous deposit.[2]

675. *Treatment.*—Although not so important a matter as in the days when profuse bloodletting was habitually recommended in acute affections, still even now the determination of the nature of tuberculous pericarditis is not a mere matter of diagnostic curiosity. The most appropriate treatment, as Dr. Burrows has successfully argued (*Med.-Chir. Trans.*, vol. xxx.), can scarcely be that adapted to rheumatic pericarditis. Bloodletting, if employed at all, should

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[1] Ryan, U. C. H., Females, vol. xiii. p. 43—the solitary example of the fact, as far as I can remember, I have met with.

[2] Mercer, U. C. H., Females, vol. xiii. p. 7.

be had recourse to in extreme moderation; mercurials must be given with all needful care to avoid ptyalism. Blisters in the neighborhood of the præcordial region, dry-cupping, moderate purgation, diuretics with iodide of potassium and alkalies, are the agents on which, in connection with antiphlogistic regimen, reliance must be placed. Animal food may be allowed earlier in this than in other varieties of the disease.

SUB-CLASS II.—GROWTHS.

676. The heart is far from ranking as a frequent seat of growths. Primary production of any one of the series within the actual muscular substance is, in truth, rare; and when we take into consideration the extensive relationship of the endocardial surface to the blood, circulating absorbed elements from primary formations elsewhere, it seems remarkable how rarely the cardiac structures become the seat even of secondary development of the kind.

677. It has not occurred to me to meet with cystoma or sarcoma singly or combined, with fibroma, enchondroma, or osteoma in the heart; and the only product belonging to the group I have actually seen, is carcinoma.

CARCINOMA.

I.—HEART.

678. Cancer of the heart is by no means so extraordinarily rare, as is commonly supposed—that is, if secondary and clinically insignificant, as well as primary and grave, examples of the disease be included.

679. Cardiac cancer may be the solitary evidence of the diathesis, or it may occur in combination with similar disease elsewhere. When thus associated, which is vastly the more common state, it is, as far as I know, always secondary.

680. Encephaloid is essentially the species found in the heart; examples of genuine scirrhus are very rare; while of colloid I have neither seen nor read of a well-defined instance. Melanic pigment is sometimes more or less copiously deposited in the stroma and within the loculi of encephaloid masses—a deposition not impressing in this locality, more than elsewhere, any special character on the cancerous substance.

The nodular is the usual form of deposition; but the sarcous substance may be infiltrated, like the muscles of the limbs, in such manner that portions of the heart seem to the eye metamorphosed into cancer. And the actual atrophy of sarcous structure is, under these circumstances, very extensive—but evidently not complete, as a case to be by and by narrated would prove, as well as a yet more remarkable one in which eleven-twelfths of the organ are said to have been involved. Had the conversion been here as complete, as it seemed, life evidently could not have held on.

Nodules may form under the endocardium and pericardium without implicating the heart's fibres—a fact of which I have elsewhere published a striking example.[1] Or they may similarly grow within the muscular substance—and in vast numbers, as the instance recorded by Dupuytren, in which he ceased to count at the six hundredth, sufficiently proves.

The part of the organ involved varies greatly; in fact every conceivable combination of localities is exemplified by actually observed cases. It does not clearly appear that the right side of the organ exhibits any notable excess of proclivity to the disease.

When the disease was nodular and secondary, I have found the heart in all respects perfectly natural; nay, even in cases of infiltration, the fibre just beyond may be thoroughly sound. There is no disposition to myocarditis, local or general; but recent endocarditis in the immediate vicinity of the cancerous formation has occasionally been found, as well as old pericarditis. The presence of dilatation or hypertrophy is purely accidental.

Cancer of the heart does not spread to other organs; but conversely extension of the disease from the mediastinum and lung to the heart is not very uncommon, where primary accumulation has occurred in those localities. Some particulars of a remarkable case of the kind are detailed a little further on. And this appears to me to be the only manner of localization of primary cancer in which the heart exhibits any particular tendency to suffer secondarily. Nevertheless, parts the most distant, the most diversified, and the most functionally disconnected with the heart, being the primary seats of the disease, the latter organ has now and then become secondarily involved—and, singularly enough, the liver and lungs, the usual seats of secondary growth, having escaped contamination.

681. Males are very notably more prone to suffer from cancer of the heart than females; of twenty-eight cases, including primary and secondary formation, three only were furnished by females. The disease occurs at all ages; if a narrative of Billard may be accepted literally, scirrhus occasionally forms in the heart even during intra-uterine life.

682. *Symptoms.*—Cancer of the heart has in the majority of recorded instances remained wholly latent. Such absence of symptoms is not to be wondered at, where minute or even pea-like nodules only have formed in the substance of the organ; but, where pretty extensive infiltration of the walls had occurred, the deficiency of sign and symptom affords fair matter for surprise.

Still such deficiency is not invariable, as the following particulars, condensed from a report by M. Andral, show. A man, aged thirty-seven at death, two years previously to that event began to suffer from slight dyspnœa. This was followed after five months by a sudden and temporary attack of severe præcordial pain, increased dyspnœa, vomiting and loss of consciousness; the succeeding year the habitual dyspnœa increased, and seven or eight paroxysmal attacks, such as just described, occurred; six weeks before death emaciation and straw-colored tint of skin were observed. In the intervals of ease nothing could be detected by auscultation. Anasarca, commencing with the lower extremities, reached the upper limbs and the face; and the patient died suddenly without struggle. Here cancerous infiltration and tumor combined had almost completely taken the place of the muscular structure of the right heart. No other morbid change, it is affirmed, could be detected in that or in any other organ.[1]

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[1] Clinical Lecture on Subcutaneous Cancers, Medical Times, August, 1852.

But there can be no surety that these symptoms were not rather the produce of obstruction of the large veins than of the change in the heart's structure. I am certain grave infiltration may exist without impressing any specific character on the symptomatology. Thus, in a case recently seen,[2] where the physical signs and a variety of concentric pressure-symptoms[3] enabled us to diagnose the existence of mediastinal tumor and left pleural effusion, the following are the notes of the examination of the heart: "Slight cardiac impulse at left costal angle, very faint in proper heart-region; no cardiac impulse or thrill to the right of the sternum or above the clavicles. . . . Heart's sounds remarkable alone for their pendulum-like rhythm; no murmur at left apex; nothing abnormal in sounds in the course of the arch of the aorta; pulse 120, excessively feeble, regular, a little fuller in left than right radial. . . . Close to the inferior angle of the left scapula is heard a strongly marked, rough, medium-pitched arterial murmur." There was no clinical evidence, then, pointing to the heart; and yet the anterior portion of the wall of the right auricle was so infiltrated with cancer as to seem metamorphosed into that substance, while the mediastinal mass pressed seriously in the direction of the limited space between the tricuspid and pulmonary orifices. The capacity of the right ventricle appears to have been reduced by the pressure of the encroaching growth. Both the left cavities were of natural extent, and the mitral valve and orifice normal. The dorsal murmur in the descending aorta was doubtless caused by direct pressure of some portion of the tumor. Immediately beyond the infiltrated substance, the muscular texture was perfectly natural.

683. The diagnosis of cardiac cancer has never, as far as I am aware, been made.

II.—ENDOCARDIUM.

684. An example of presumed cancerous ulceration of the endocardium has been recorded by M. Payan.[4] The details given appear

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[1] Anat. Pathol., t. ii. p. 328. 1829.

[2] Scammell, U. C. H., Males, vol. xvii. p. 275; and Univ. Coll. Museum, No. 4590.

[3] Diseases of Lungs, 3d Am. edit., p. 152.

[4] Revue Médicale, Nov. 1841.

to me anything but conclusive as to the genuinely cancerous nature of the ulcerations described. Nor is there any proof that the encephaloid mass found by M. Hudellet attached to the wall of the right auricle, almost filling its cavity, and extending into the ventricle and both venæ cavæ, was really produced from the endocardial surface. Probably cancer-elements underwent evolution in an enlarging blood-coagulum. The primary disease lay in the parotid glands. No chest symptom, singular to relate, had existed; death occurred instantaneously after a meal.[1]

III.—PERICARDIUM.

685. Minute cancerous nodules occasionally form under the serous membrane, both parietal and cardiac. But, even if they acquire some size, they will not only fail to produce symptoms, but will cause no friction-murmur, unless the serous surfaces be rough.[2]

A case of accumulation of cancerous substance in layers within the serous sac, recorded by Dr. Bright,[3] is rendered peculiarly interesting by the occurrence of aggravated attacks of paroxysmal dyspnœa, mainly taking place by night, and accompanied with general agitation and spasm of the diaphragm. These seizures were probably direct and reflex results of irritation of the phrenic nerve imbedded in the morbid mass.

Sub-class III.—Pseudo-Tissues.

Induration-Matter.

CIRRHOSIS.

686. Infiltration of the connective tissue and muscular interspaces of the heart with exudation, which hardens into induration-matter of low plastic and contractile type, is a very real, though a rare, morbid condition of the organ.

Assimilable in nature to cirrhotic infiltration in the liver, kidney and lung, in the heart also the induration-matter is laid down, in sequence to congestion, and not to an actively inflammatory process. But, whereas the former organs are very commonly affected on an extensive scale, if affected at all, limited portions only of the cardiac substance undergo the change—either an islet of the ventricular walls, or some portion of the columnæ carneæ, especially the papillary muscles.

The infiltrated material is sometimes structureless, sometimes obscurely fibroid. It may undergo fatty metamorphosis, or become calcified, or even pseudo-ossified. I have seen the latter condition of a papillary muscle attended with the like alteration in some of the attached tendinous cords. Atrophy of the proper muscular texture proceeds *pari passu*.

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[1] Cruveilhier, Anat. Pathol., Livraison xxix.

[2] Clin. Lect. on Multiple Subcutaneous Cancers, Med. Times, 1852.

[3] Med.-Chir. Trans., 1839.

The inherent disposition of induration-matter to contract is, in the wall of the ventricle, controlled and overcome by the eccentric pressure of the blood; and the resistant power of the material being small, the result is local distension. True sacculated aneurism of the heart originates more frequently in this than in any other manner. Rupture of the dilated part of the wall of the heart may eventually occur.

687. Although commonly traceable to passive, sometimes to mechanical, congestion, this cirrhotic state of the heart's texture occasionally occurs independently of any perverted state of local circulation. At least no sort of evidence can be found in some cases, either in the past history or the *post-mortem* appearances, warranting a belief in such perverted state.

688. This alteration is excessively rare in the walls of the ventricles, and almost confined to the left; but a greater or less degree of the change is not very uncommon in the papillary muscles. Now in the former situation it is not revealed by either symptoms or signs, unless it have proceeded to such lengths as to have induced aneurismal pouching, of which the clinical effects, elsewhere described, will prove more or less obvious. In the latter situation the infiltration must of necessity, if it have proceeded to contraction of the papillary muscles, entail imperfect closure of the mitral orifice with the murmur distinctive of that state.

CLASS III.—PARASITES.

ENTOZOA.

689. The heart occasionally affords a nidus to cysticerci, and to acephalocysts. I am not aware that the trichina spiralis has ever been found in its substance.

690. It would appear that multiple broods are rare in the acephalocysts of the heart—generally, indeed, the mother-cyst is solitary. In this organ, as elsewhere, the cysts afford a habitat to the echinococcus. Sometimes acephalocysts have coexisted in their common seat, the liver; sometimes the heart alone has been affected.

691. The septum of the ventricles appears to be a favorite cardiac seat of these entozoa.

692. Symptoms do not occur as matter of necessity: their absence or presence will depend on the mechanical influences exercised by the sac. Thus among Sir R. Carswell's unpublished drawings is the figure of a heart containing in the posterior part of the wall of the left ventricle a good-sized acephalocystic sac, protruding somewhat on the pericardial surface.[1] The patient, a female, was cut off by phthisis, and the sac accidentally found; and as she had died in hospital, and the heart had not attracted attention, the probability is there were no cardiac symptoms.

On the other hand sudden death may be caused by the mechanical influence of these productions. Thus I have before me a heart containing an acephalocystic sac, as large as a pigeon's egg, in the septum of the ventricles:[1] the patient, a female, dropped dead suddenly, while engaged in her household affairs. Professor Rokitansky relates a singular case in which an acephalocyst, nearly as large as a hen's egg, escaped from the mother sac in the upper part of the ventricular septum, and caused instant death by becoming impacted in the conus arteriosus and pulmonary artery—reaching almost to the left branch of this vessel.

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[1] Univ. Coll. Mus., A. 9.

§ VIII.—DISEASES OF THE ORIFICES AND APPENDAGES.

693. Diseases of the orifices of the heart, though not wholly unproductive of pain and other forms of subjective inconvenience, are essentially injurious through their mechanical effects. They interfere in either, or both, of two ways with the circulation through the organ. They are constrictive in anatomical character, and hence produce *obstruction* of the onward current: or they entail disproportion between the size of the orifice to be closed and the valve to close it; or they interfere with the play of the closing apparatus, and in either of these two ways lead to *reflux* or *regurgitation* of a backward current. In all these cases impediment to the onward-moving stream is the result.

694. Each orifice is in theory capable of each kind of disease—obstructive and regurgitant; but the frequency with which the various orifices actually suffer differs very widely. This question, however, requires to be considered in two points of view, the anatomical and the clinical: the excess of organic change in the actual texture of the valves and orifices of the left side, is very great; the excess of disturbance, clinically demonstrable, in the functional conditions of the orifices of the left side, much less. This comes of the frequency with which the tricuspid valve is incompetent, through mere widening, without textural change, of the orifice, while its own structure remains sound: the excess of textural disease of the valves on the left side is counter-balanced by the excess of misfitting of one of the closing valves on the right.

695. Obstruction of orifices, as far as is known positively, is always of organic character, and is caused by morbid change in the valves, in the wall of the orifice itself, or by extraneous pressure. Regurgitation through orifices is, certainly, in the vast majority of cases, likewise of organic character, and is caused by morbid change in the valve, in the wall of the orifice, or in some part of the apparatus connected with the closure of the valves. But it is next to certain that regurgitation may be produced

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[1] Univ. Coll. Mus., No. 2293.

dynamically in the mitral orifice at least, through functional imperfection of the closing apparatus, though this be perfectly free from organic change: regurgitation of this kind is, however, of temporary duration, and does not produce results of importance. It is possible, too, that obstructive disturbance may be produced at the mitral orifice by perverted action of the papillary muscles; and either regurgitation or obstruction at the mouth of the aorta by perverted innervation of the sigmoid valves: but all this is completely hypothetical at the present day; and hence, practically, the serious diseases of orifices may be regarded as organic and statical.

696. Diseased valves, diseased orifices, and diseased states of the apparatus effecting the closure of the valves, are, then, the causes of perverted circulation through the outlets, and through the channels of communication between the cavities, of the heart.

(a.) *Diseased valves* are the causes of *obstruction:* first, through local endocarditis and its products interstitial and superficial, alterations of form, and adhesions of the divisions of the valves *inter se;* secondly, through deposits of fibrin from the blood on their surfaces; thirdly, through atheroma, calcification and their sequences; and, fourthly, in some instances, through hypertrophy. Diseased valves, again, are the source of *regurgitation:* first, through endocarditis and its effects (especially when these tend to produce puckering and diminished superficial size, or actual destruction of the divisions of the valves, or adhesion of these either to each other, or to the adjacent wall of the heart or great vessel to which they belong, or to entail thickening and shortening of the chordæ tendineæ, or rupture of these); secondly, through fibrin deposited among, and interfering with, the action of the chordæ tendineæ; thirdly, through atheroma and its sequences; fourthly, through hypertrophy of the valves interfering with the freedom and completeness of their fall at the proper moment; fifthly, through atrophy of the valves, affecting their depth or superficial extent, in rare instances affecting their continuity by extensive perforation; and, sixthly, through atrophous shortening of the chordæ tendineæ.

Certain diseases of valves may act now as the cause of regurgitation, now as that of obstruction, functionally considered. In a remarkable case of the kind where the time of a murmur at and above the left apex had during life puzzled exceedingly those who watched the case from day to day, inasmuch as that murmur appeared purely systolic at one time, at another post-systolic, and again systolic and diastolic both, the explanation clearly arose out of the state of the mitral valve. The long division of that valve exhibited a rounded perforation, as large as a good-sized pea, closed up by a soft fibrinous vegetation, attached so loosely, however, to the edge of the opening, that the two things—perforation and plug—must have occasionally changed their relationships in such manner that either obstruction to, or regurgitation of, the blood-current, or both, must have been the passing results.[1]

(*b*.) *Diseased orifices*, the actual substance of the valves being sound, lead to *obstruction* through the products of endocarditis, atheroma, and calcification lying upon, or producing changes in, their surfaces or walls. And disease of the orifices is the cause of *regurgitation*, where the valve being sound, the opening is morbidly widened to such an extent as to render the former inadequate to its closure.

(*c*.) *Diseased states of the apparatus*, connected with the closure of the valves, are seen in induration and cirrhosis with diminished bulk of the papillary muscles, and in shortening of the chordæ tendineæ; they produce *regurgitation*. I once met with a direct mitral murmur, where the orifice, instead of being obstructed, was actually too wide; a chorda tendinea from the long tongue of the valve bifurcated, each division attaching itself to a separate papillary muscle, and so placing a thread-like obstruction in the way of the current.[2] It may be made a question, too, whether imperfect action of the papillary muscles, from textural disease of their substance, may not act obstructively also, and throw an obstacle in the way of the passage of the blood from the auricle to the ventricle.

697. Such are the general characters and immediate effects of the morbid states of the orifices and their connected apparatus. We will now pass to the consideration of the signs and symptoms of these morbid states at each orifice. These signs and symptoms, it must be noticed, are essentially of mechanical origin; and hence their discovery discloses the existence simply of obstruction or regurgitation at a certain orifice, but tells nothing directly, and by necessary inference, as to the nature of the morbid process, whether inflammatory, fatty, calcifying, fibrillating, exudative, ulcerative, or cancerous, that has engendered the mechanical difficulty. In the majority of cases valvular disease entails some form of anatomical change in the substance of the heart itself: the proper signs of the former and of the latter are, therefore, met in frequent clinical association; for purposes of precision, it is necessary each class of signs should be enumerated apart—as is done in the following descriptions. As already shown, too [219], different valvular affections often coexist, and in certain combinations the one may tend to throw into the shade, or actually modify, the signs of the other or others.

698. To save the necessity for repetition with the account of each valvular disease, it may be stated here, once for all, that valvular disease *per se* never alters the area or intensity of the heart's dul-

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[1] Goodwin, U. C. H., Males, vol. xi. pp. 41, 85; further illustrated clinically by case of Williams, U. C. H., Males, vol. xvi. p. 10.

[2] Pickett, U. C. H., Males, vol. vi. p. 231.

nesses, superficial or deep-seated. If either be affected, the presence of some other morbid state becomes matter of certainty.

MITRAL REGURGITATION, REFLUX, OR INSUFFICIENCY.

699. How is the reality of mitral regurgitation during life to be demonstrated after death? Tie the aorta and coronary arteries, cut off the apex of the heart, and let fall against the mitral valve a full-sized column of water; the valve will either support the column, or allow it to pass through to the auricle. If the whole of the ventricle above the floor formed by the valve remain filled with the water, scarcely a drop filtering through, there can be no doubt the valve was competent during life.[1] If, on the other hand, the water escape rapidly, the incompetence of the valve may be held certain. But in the case of slight filtration through the orifice, it would scarcely be justifiable to infer the insufficiency of the valve during life, as we have no means of imitating the vital contractions that accompany the act of closure in the living and moving heart.

700. *Physical signs proper.*—The impulse, in highly marked disease of this species, is, in the majority of cases, irregular in force and rhythm. Systolic thrill may sometimes be felt at and about the left apex.

But the essential character of this regurgitation is systolic murmur of maximum force at the left apex, and possessing the other characters already enumerated [211]. The systolic sound, completely or incompletely covered in this position by the murmur, may be perfectly natural at the ensiform cartilage and at the mid-sternal base; the second sound, often weakened, in consequence of diminished calibre of the aorta, at the second right, or aortic, cartilage, is accentuated in many, but not in all, instances at the second left, or pulmonary, cartilage. Sometimes the second sound is distinct and sharp at the left apex, much sharper than at the pulmonary cartilage; a fact illustrated by the case (Anne Gippin) quoted a little further on.

Systolic murmur limited to the left apex is never of blood origin, as far as I have observed, therefore never chlorotic: the existence of chorea will distinguish the peculiar dynamic murmur of that disease: the murmur of this site and rhythm attending simple dilated hypertrophy will commonly disappear under treatment. The distinction of murmur caused by mitral regurgitation and that produced by friction of blood against irregularities of surface at the base of the ventricle, cannot, I think, be made with positiveness in the present state of knowledge—but fortunately the latter kind of mechanism is excessively rare. I have already spoken of the influence of feebleness of the heart in preventing the evolution of murmur, of which the physical causes exist: when the heart is texturally feeble, as in fatty softening, its action may be excited by a few turns in a room, or by any other effort, and the murmur will then become audible; when it is feeble from collapse, this must be allowed to pass away, before a diagnosis is ventured on.

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[1] Such was the fact in Wilkinson, U. C. H., Males, vol. ix. p. 306; Landers, U. C. H., Females, vol. ix. p. 289; Fosbury, U. C. H., Males, vol. x. p. 133; Doyle, U. C. H., Males, vol. x. p. 238; Snail, U. C. H., Females, vol. x. p. 260.

701. *Indirect Physical signs.*—Dilated hypertrophy of the left ventricle is the common sequence of mitral regurgitation; hence the apex-beat and impulse generally are carried outwards, and lowered somewhat. The impulse is increased in force, and in rare instances there is auricular impulse at the second interspace—either pre-systolic, when it comes of hypertrophy of the auricle, or systolic, when it is communicated from the ventricle. The area of percussion-dulness is increased, especially to the left: but, from sequential hypertrophy of the right ventricle, which I have oftenest observed in early youth, extension of dulness may eventually take place to the right also.

702. *Pulse.*—The pulse presents itself in either of the two following states: Regular in force and rhythm; small, with occasional sharpness; rather frequent, and compressible, unless there be much hypertrophy: or irregular in force and rhythm, sometimes to an excessive degree, small, feeble, with occasional sharpness, and tremulous under excitement of the heart. In either of these states, the systoles and radial pulses may fail to correspond in rhythm and in number.

703. *Effects on the Capillary systems.*—Pure mitral regurgitation produces but little effect on the systemic capillary circulation. The disease may exist for years without inducing either general dropsy or systemic congestion. If dilated hypertrophy supervene, systemic obstruction occurs with a facility proportional to the amount of dilatation and, especially, the impoverishment of the blood. The symptoms then become those of dilated hypertrophy, aggravated, *plus* certain others, to be by and by mentioned, more or less peculiar to the regurgitation. I use the word "aggravated" advisedly; it has not occurred to me to observe cases justifying the idea that mitral regurgitation acts as an efficient safety-valve to a dilated and hypertrophous left ventricle.

The disturbed conditions of the sensorial functions sometimes observed in mitral disease are likewise the results rather of the dilated hypertrophy with which it is associated, than of itself alone; still, when carried to a great amount, it may secondarily, through its influence on the pulmonary circulation and the right heart, tend to congest the brain passively. No cases have fallen under my notice supporting the notion that colorless softening of the brain is a direct dependence on mitral regurgitation—a notion very ingeniously advocated by Dr. Law.

The essential effects of mitral disease are pulmonary—congestive and irritative. The current thrown back through the auricle on the pulmonary veins tends to congest the lungs; and it is not unreasonable to admit that the effort on the part of the right ventricle to overcome the increasing obstruction may induce irritation. Cough, watery expectoration, dyspnœa, and orthopnœa; actual bronchitis, pulmonary œdema, pneumonia, either congestive or irritative, passive congestion and pulmonary apoplexy, are the direct effects of the disease. The expectoration may by the latter condition be stained of dark blood tint, or blackish; or actual escape of pure blood may occur: what maximum quantity of blood may be brought up from this cause at a time, I do not know; I have never seen any large amount.

MITRAL OBSTRUCTION.

704. *Physical signs proper.*—I have never observed diastolic thrill at the left apex, though it is conceivable that, if a highly hypertrophous left auricle lie behind the constricted orifice, the current may be rendered sufficiently strong to produce that thrill [85]. The impulse is irregular and unequal in force. The characters of the murmur of mitral obstruction will be found elsewhere [215]; its frequent absence renders the positive diagnosis of this form of disease far from easy. I have already stated that the rhythm of the murmur is rather post-diastolic or præ-systolic, than actually diastolic: I have met with no satisfactory proof of its ever being systolic, as has been affirmed in some quarters—the evidence given, in such alleged instances of systolic constrictive murmur, to show the absence of regurgitation, being besides, in my mind, insufficient.

Even where a hypertrophous auricle lies behind a constricted mitral orifice, no murmur necessarily ensues, if the constricted surface be smooth, nay, even if it be rough—this is proved by the following excerpt:—

> "Anne Gippin, ætat. 35. . . . p. = 144, regular in rhythm, irregular in force, feeble, small; r. = 48; anasarca; ascites; jugulars swollen, pulsatory; radials faintly visible at wrist; heart's action too extensive—impulse slapping rather than heaving; faint thrill at apex-region. At mid-sternal base both sounds dull; first slightly murmur-like, and accompanied by knock; at the left apex systolic roughish murmur; while the second sound is distinct and much sharper than at the pulmonary cartilage. . . . . *Post-mortem* (twenty days later): heart, with 1½ inch of great vessels, weighs 13¼ oz; tricuspid orifice measures 4⅞ inches; pulmonary orifice measures 3⅜ inches; the tricuspid valves looked insufficient to fill the widened orifice, but no experiment done; left auricle very hypertrophous, in some parts wall = ¼ inch in thickness, its endocardium creaks in cutting; mitral orifice just admits end of index-finger, measures 1⅞ inch; edge is rugose, and valve is directed in funnel shape towards the ventricle; chordæ tendineæ, very thick, are so shortened that practically the papillary muscles seem inserted directly into the valves; aortic orifice, water passes gradually through from above (but the coronary arteries were not tied); fine warty yellow vegetations, especially on corpora Arantii; width of orifice = 2⅜ inches." . . . . (U. C. H., Females, vol. v. pp. 292–299, Dec. 1850.)

Here the second sound was clearly defined at the left apex, there being no constrictive murmur; but that sound may be audible there, even when mitral constrictive murmur actually exists. On the other hand, so frequent is the association of regurgitation, that it is very rare to find a pure first sound at that point. At the mid-sternal base the first and second sounds are of natural character, except that they are sometimes feeble, from the smallness of the current sent on from the left ventricle. At the aortic cartilage, both sounds are feeble; at the pulmonary, both, on the contrary, and especially the second, are full and accentuated: if there be coexistent insufficiency of the tricuspid valve, this accentuation may, however, as in the case just referred to, be prevented from occurring.

705. *Indirect Physical signs.*—Left auricular præ-systolic impulse, and right ventricular impulse, are frequently found as coexistences, in consequence of dilated hypertrophy being gradually produced in those situations. The pathological reasons of these enlargements are sufficiently clear. The area of percussion-dulness, especially to the right side, will on similar grounds be increased.

706. *Pulse and Capillary systems.*—The character of the pulse and the pulmonary symptoms are the same as in regurgitant disease of this orifice.

When constriction and regurgitation coexist, as is commonly the case where there is constriction, either the murmur of regurgitation is heard alone, or a double to-and-fro murmur is distinguished; its two portions differing in quality and pitch.

Skoda, however, appears to hold that when the two physical imperfections exist, there are always two attendant murmurs—the apparent singleness of murmur in some cases being an illusion dependent on the two murmurs following each other so closely as to simulate continuousness, while the quality and pitch of both are indistinguishable. I cannot help thinking that, on the contrary, in these cases the ear catches solely the systolic murmur, which is prolonged through, and covers the weaker murmur of, diastolic time.

TRICUSPID REGURGITATION, REFLUX, OR INSUFFICIENCY.

707. *Physical signs proper.*—I have never known tricuspid regurgitation productive of thrill; the characters of its attendant murmur, which, for reasons there assigned, is often absent, are set down in a previous place [212].

The second sound is habitually weak at the right apex, almost to extinction sometimes—in all probability in consequence of the smallness of the current sent on through the pulmonary artery. In an instance of well-marked tricuspid regurgitation, where the second sound was exceptionally strong at the right apex, the peculiarity seemed traceable to dilatation of the pulmonary artery just above the sigmoid valves.[1] Both sounds at the base and at the aortic cartilage may retain their natural characters; if the disease be pure, the second sound is weak at the pulmonary cartilage.

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[1] Hishin, U. C. H., Females, vol. xi. p. 286, or Med. Times and Gaz., Feb. 14, 1857.

708. *Indirect Physical signs.*—Commonly coincident with dilatation, simple or hypertrophous, of the right ventricle, the signs of these conditions are discernible:—Epigastric and right sternal impulse, of variable force, but out of proportion with that on the left side, unless the left ventricle be accidentally hypertrophous also, and increased area of percussion-dulness, mainly to the right. Hypertrophy of the right ventricle will prevent the enfeebling of the second sound appertaining to the pure regurgitation.

709. *Pulse.*—Those arteries, the pulmonary, which tricuspid insufficiency may be supposed to affect directly, cannot be felt; and there is nothing peculiar in the pulse of the branches derived from the aorta. The cervical veins distended, knotty, pulsatile, and refilling readily from below, may be the seat of thrill. But all these various signs are only found prominently in well-marked cases; if the regurgitation be slight, or if the right ventricle be very weak, venous pulsation, and thrill especially, may be altogether absent. Nay, even if the right ventricle, and especially its infundibulum, be strong, these signs may be wanting.[1]

710. *Effects on the Capillary systems.*—The pulmonary capillaries not only escape primary congestion in this form of disease, but may be considered theoretically to be under-supplied with blood. Be this as it will, however, they do in the course of time undergo secondary engorgement in sequence to systemic obstruction. That the systemic capillaries are, in truth, the primary and immediate sufferers, has been very satisfactorily shown by Dr. Blakiston. The student must, however, bear in mind that the relationship between tricuspid regurgitation and grave systemic stagnation is not so intimate and essential that, given the former, he may take it for granted the latter must, *e necessitate rerum*, follow [617, 9]. And I further believe that the concomitant dilatation of the right cavities plays a very essential part in generating stagnation and dropsy, where these actually occur. The systemic effects are the same as those occurring in dilatation, and in the section on that disease their description will be found.

Dr. Blakiston's results tend powerfully to show that obstruction of the cerebral capillary vessels and apoplexy are much more frequent in cases of heart disease with, than without, stagnation of the systemic capillary circulation: hence the inference, that cerebral obstruction and apoplexy are more connected with the heart through general cardiac dilatation and tricuspid regurgitation than through hypertrophy of the left ventricle.

Again, the conclusion has been suggested to me by a small number of facts, that tricuspid regurgitation plays a certain part in generating cirrhosis of the liver—difficult though it may be to readily understand how the necessary influence is exercised through obstruction in the circulation of the hepatic veins. That jaundice is sometimes induced through such obstruction, is indubitable.

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[1] Hishin, loc cit.

Congestive albuminuria, as also engorgement and œdema of the female pudenda, with passive leucorrhœa, is much more frequently met in the present than in any other condition of valvular disease.

711. It may on first thought appear strange that such serious consequences should be ascribed to tricuspid regurgitation—a phenomenon which the surmise of John Hunter, the arguments of Mr. Adams, and the experiments of Mr. King, would lead us to suppose exists in health, as a normal provision against overloading of the pulmonary capillaries.

But does such normal regurgitation really exist? Valentin denies it.[1] Dr. Hope conceived that the absence of tricuspid regurgitant murmur in healthy persons sufficiently disproved the possibility of any such regurgitation: his argument was, however, valueless, for, as is well known, even morbid regurgitation, highly-marked, may exist without any murmur. Still I agree with Dr. Hope in his refusal to admit the reality of regurgitation in health: my grounds are, that if regurgitation existed, it would visibly affect the venous circulation in the neck,[2] and that the experiments on which the doctrine is founded do not represent what common sense leads us to suppose must be the state of action at the tricuspid orifice during life. Besides, if, as is the fact, the valve, provided itself and the orifice be perfectly natural, will, after death, support water poured into the ventricle from the apex, how can we admit that during life the mechanical action shall be less complete? Conceding, however, *argumenti gratiâ*, that slight and occasional regurgitation is a normal fact, it might nevertheless be true, as actually is the case, that highly marked and permanent regurgitation should act as the source of most serious disorder.

TRICUSPID OBSTRUCTION.

712. *Physical signs proper.*—Constriction of the tricuspid orifice would theoretically give a diastolic murmur of maximum force at the ensiform cartilage. But this condition of the orifice is excessively rare, because atheroma and calcification are themselves extremely uncommon in this situation; and an amount of coarctation reducing the orifice to the size of the middle finger may not, as a case observed by Dr. Hope proves, give rise to murmur—probably on account of the weakness of the current.

713. The following extract from my case-books seems to furnish the characters of tricuspid obstruction and regurgitation with preciseness, but there was no *post-mortem* examination to settle the point decisively:—

"Wm. Hodson, ætat. 48; anasarca; constant cough, with rather profuse muco-purulent expectoration; no albumen in urine. Heart's apex beats feebly

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[1] Physiologie, Bd. i., S. 425.

[2] The case of Hishin above referred to [709], however, makes me now doubt the real justness of this argument.

in fifth space; its deep-seated dulness does not reach more than an inch to the right of the sternum. Both external jugulars considerably swollen, the right highly pulsatile from below, the left less so, though a small communicating vein, crossing the clavicle, pulsates slightly. At mitral apex, no murmur; first sound feeble, second sound clear, full and accentuated. At tricuspid apex, that is precisely at point of left costal angle [12], there is a soft double murmur, the diastolic division much better marked about level of the fourth interspace. At the mid-sternal base, just below the level of the third cartilages, it cannot be said that there is any murmur. The second sound at the tricuspid apex is frequently reduplicate, very rarely so at the mid-sternal base: the diastolic-timed murmur is louder and more prolonged than the systolic. Pulse visible in posterior tibials and radials, not in femorals. No cervical hum. . . . . . .
In sitting posture second sound almost constantly reduplicate at tricuspid apex, whereas in at least twenty beats only once reduplicate at base, and not once at mitral apex." (U. C. H., Males, vol. ix. pp. 64–67.)

The heart not having been lowered in position, it would appear unjustifiable to refer the diastolic murmur, heard at the left costal angle, to aortic regurgitation.

AORTIC OBSTRUCTION.

714. *Physical signs proper.*—In well-marked cases, systolic basic thrill may be caught; and the murmur already described [213] exists. The natural first sound of the heart remains audible at the left apex, though more or less covered by the basic murmur: the second sound, weak at the apex, at the base, and at the aortic cartilage, may be slightly murmurish; at the two latter points, in consequence of some regurgitant tendency. The second sound is occasionally reduplicate at the base. The systolic murmur may be prolonged enough to cover the second sound more or less completely at the base; the latter may then, as a rule, be caught at the apex.

The murmur of chronic aortic constriction is one of those open to the greatest number of imitations. Of acute endocarditis, pressure by pericardial fluid or false membrane, as causes of similar murmur, I have already spoken; and, further, the distinction of this murmur from that produced by disease of the aorta close to the valves, and by abnormal communications within the heart, is far from easy. It has already been shown that displacement or twisting of the heart on its axis does not necessarily produce murmur, though this sometimes occurs. Of the blood-changes, producing systolic basic murmur, spanæmia is the most important; its distinctive characters have already been inquired into.

Weakness of action of the heart, or extreme smoothness of the constricted orifice, may prevent the development of murmur.

715. *Indirect Physical signs.*—The impulse and altered percussion-sound characteristic of hypertrophy, with or without dilatation of the left ventricle, are the commonly coincident signs. A rare instance every now and then occurs, wherein the murmur of mitral insufficiency is generated through dilatation of that orifice following

on dilatation of the ventricle, which, in turn, has been entailed by the enduring obstruction at the mouth of the aorta.

716. *Pulse.*—The pulse, in cases of moderate coarctation, is not materially affected. If the constriction be great, the pulse, though regular in force and rhythm, is small, hard, rigid, concentrated; hardness and force signify hypertrophy behind the narrowed orifice.

717. *Effects on the Capillary systems.*—Aortic constriction exercises no direct effect on the pulmonary capillaries; some indirect tendency to stagnation arises from the difficulty experienced by the blood, flowing from the pulmonary veins, in its entrance into a ventricle, which, again, has itself great difficulty in discharging its contents.

It is truly remarkable to what an extent this coarctation may be carried, without producing systemic stagnation: the opening may be no larger than a pea, without leading to the very slightest œdema, even of the ankles. This immunity, however, only holds as long as the capacity of the ventricles and the width of the tricuspid orifice remain unaffected; if the blood becomes spanæmic, too, anasarca occurs independently of these latter changes. It has been conjectured that the peculiarity depends on the slowness of the circulation in old people; but the circulation is not always slow in old people, and young adults, with constriction of the aortic orifice, also remain free from systemic dropsy.

It seems theoretically probable that a constricted aortic orifice will weaken the impulsive effect of a hypertrophous left ventricle on the brain.

AORTIC REGURGITATION, REFLUX, OR INSUFFICIENCY.

718. *Physical signs proper.*—Diastolic basic thrill, though it has not fallen under my notice, may conceivably be perceptible, especially if the blood be at all spanæmic. I refer to a previous place [217] for the characters of the murmur of aortic insufficiency. The state of the heart's sounds is as follows: The first, at the base, may be natural or very nearly so, dull, obscured by a soft murmur, or masked totally by a harsh one. The second sound, at the left apex, may be null, faint, or distinct and sharp; in the latter case, the sound heard is either that of the pulmonary valves transmitted, or possibly that of fall of the aortic blood, during regurgitation, into the ventricle below [149[1]]. At the aortic cartilage the first sound is of variable character; the second, a murmur, more or less marked. Both sounds may be perfectly natural at the pulmonary cartilage, and occasionally the second accentuated, though there be no mitral regurgitation: occasionally, however, the basic dias-

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[1] In addition to what is said, in the place referred to, on the possible generation of an imitation of natural second sound by fall of blood from the aorta against the inner surface of the apex of the ventricle, it occurs to me to suggest whether, if the phenomenon be ever really sonorous, the noise is not more likely to possess the characters of a murmur than of the diastolic sound of the heart.

tolic murmur is very loudly conducted to this cartilage, covers the second sound there, and *pro tanto* simulates the essential sign of insufficiency of the pulmonary valves.

Aortic regurgitant murmur is, as far as I know, constant, where its physical cause exists; weakness of ventricular action obviously cannot have the same effect in rendering a regurgitant, as a direct, murmur obscure. If a double aortic murmur exist, the systolic portion is best transmitted upwards in the course of the vessel, the diastolic generally downwards in the line of the sternum—the latter peculiarity probably owing to the direction of the current. The infrequency of direct tricuspid murmur and the co-existence of other signs of aortic regurgitation, will commonly prevent any error arising from the loudness of the aortic murmur at the ensiform cartilage [713]. It is conceivable that pressure on the outer surface of the aorta just above the valves, might interfere with the closure of these, and so produce regurgitant murmur; thus in a case of displacement of the heart by pleuritic effusion, diastolic murmur existed under circumstances tending very positively to connect the murmur with the displacement.[1] I have never met with a positive example of aortic *blood-murmur* diastolic in time, but such a murmur may be simulated in the following way:—spanæmia exists, with a strong systolic basic murmur; at the same time, deep-seated hum is present in the pulmonary veins; this hum is covered during the systole by the strong, blowing aortic murmur, but becomes audible during the diastole, when there is no aortic murmur to interfere with it.

Though murmur be always present in aortic insufficiency, it may certainly be masked. Thus, in a case of aortic constriction, with systolic basic murmur, and with superficial pulses visible in the highest degree, the systolic murmur was so prolonged as to cover completely any diastolic murmur that may have really existed: but the diagnosis of aortic insufficiency, made on the ground of the extreme visibleness of the pulses, was proved to be perfectly correct at the *post-mortem* examination. I can, as a bare possibility, conceive complete destruction of the aortic valves, with smooth surface, to exist without murmur; but have never observed the fact.

719. *Indirect Physical signs.*—The concomitant signs are those of hypertrophy, eccentric or simple, of the left ventricle; frequently of aortic constriction, and occasionally of mitral regurgitation.

720. *Pulse.*—The pulse is sudden, abrupt, short, jerking—a distinct fall back following instantly the rise of the vessel. Commonly regular in rhythm, sometimes notably lagging behind the systolic sound of the heart [172], occasionally bisferiens, each pulsation is, in rare instances, attended with thrill. Thrill may be powerful in the subclavian artery, very feeble in the radial, indeed only producible by artifice [258]. The amount of significance of visible-

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[1] H. Morris, U. C. H., Males, vol. vi. p. 35.

ness and locomotion of the superficial pulses has already been discussed [251]. It may be added that aneurismal varix of the aorta and pulmonary artery will likewise produce such visibleness.

721. *Effects on the Capillary systems.*—Aortic regurgitation affects the pulmonary circulation, either indirectly through mitral regurgitation (of which it is itself the original cause), or more directly through the embarrassment produced by the collision of blood, falling backward from the aorta, with blood coming forward from the auricle. The tendency to sequential hypertrophy of the left ventricle, which sometimes arises mainly about the apex in these cases, does not, as in aortic constriction, afford any help to the circulation; on the contrary by increasing the amount of distension of the aorta at each ventricular systole, it intensifies the force of the succeeding recoil.

The cerebral capillaries can only be affected secondarily through the pulmonary class; and the systemic capillaries are much in the same position. Regurgitation may exist to the highest amount without œdema of the extremities.[1]

PULMONARY OBSTRUCTION.

722. *Physical signs proper.*—Constriction of the pulmonary orifice exists with some frequency in cases of cyanosis, but coupled with such other conditions as render the attempt to fix on it special signs and effects difficult almost to impossibility. On the other hand, isolated constriction of the pulmonary orifice is necessarily rare, in consequence of the rarity of atheromatous and other changes in its valves. The characters of the attendant murmur will be found elsewhere [214]. Dr. Hope teaches that a pulmonary constrictive murmur seems closer to the ear than an aortic, and on a "higher key, ranging from the sound of a whispered *r* towards that of an *s*;" the reason being that the pulmonary artery is nearer to the surface of the chest than the aorta. As regards closeness to the ear, his statement is, doubtless, correct; as regards higher pitch, the reason assigned for this is obviously erroneous; and, indeed, Dr. Hope himself unconsciously admits this, by adding that he has known the murmur "fall below *r*, when the circulation was feeble and slow and the obstruction slight."[2]

723. *Indirect effects.*—It is difficult to conceive that pulmonary constriction can exist to any extent without inducing dilatation,

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[1] *E. g.*, Barnes, U. C. H., Males, vol. v. p. 216; but the fact is a common one: I have known this absence of dropsy, where the signs of regurgitation had existed for at least three years.

[2] Of three cases, given by Hope, in illustration of the murmurs of pulmonary constriction and regurgitation, one only terminated fatally, and here (Op. cit., p. 598) the notes only state "there was a murmur over the semilunar valves." Hence his volume gives no positive information on the subject. That a systolic murmur in an anæmic woman is louder at the pulmonary than the aortic cartilage, does not by any means prove, as he appears to think it does, that the orifice of the aorta is not the seat of the murmur.

with more or less hypertrophy, of the right ventricle, and systemic venous obstruction of a mechanical character. The pulse will probably present no uniform attributes.

PULMONARY REGURGITATION, REFLUX, OR INSUFFICIENCY.

724. The theoretical characters of the murmur, significant of this state, have been already given [218]. No jerking character of the pulse, nor visibleness of the superficial pulses of the limbs would accompany a basic diastolic murmur of this origin. By a singular fatality, while a certain number of examples of such destructive disease or insufficiency of the valves, as must have led to full regurgitation, have been observed *post mortem* in this country, in but one, that I know of, have the physical signs been clinically established. Theoretically, the effects on the systemic and cerebral capillary circulation must be most serious; and a sensation of dyspnoea, arising from the smallness of the quantity of blood actually reaching the lungs by each systole, might, unless the force of habit would counteract this influence, be expected.

725. The case referred to, observed by Dr. Gordon, is to be found in Dr. Stokes' recent volume. A boy, aged twelve, admitted into hospital with cold surface, feeble palpitation, very weak pulse, cough, copious expectoration and moist rhonchus, had thrill over the entire cardiac region, and a double murmur, loudest at the base, and inaudible at the apex, without murmur, fremitus, or visible pulsation of the superficial arteries. The thrill and "anomalous circumstances of the case" led Dr. Gordon to diagnose open foramen ovale: he was right—a large opening existed between the auricles. But the pulmonary valves, thickened and shortened, allowed a column of water to pass through; the other valves were generally healthy.

Unfortunately the rhythm of the thrill is not mentioned. It would be opposed to our experience of the parallel aortic affection, to suppose thrill produced by pulmonary regurgitation; the sign probably depended on the obstructive influence of the diseased pulmonary valves, and would have been systolic in time.

726. A loud musical diastolic murmur (where and in what direction most distinctly audible is not mentioned) was noted by Dr. Stokes, in a case of incompetence of the pulmonary valves, caused by widening of the orifice, the valves being sound: there was no aortic insufficiency.

727. *Physical Diagnosis of Valvular Diseases in general.*—In fixing on the seat of production of any given murmur, the first point for the observer to establish is the position of the heart itself: if this be abnormal, as it very often is in consequence of changes in its substance, preceding, coadvancing with, or sequential to the existing valvular disease, allowance must be made for the influence of such displacement in altering the maximum position of murmurs.

Now, the general tendency is to some form of enlargement of the organ; and, as enlargements lower it, the maxima points of murmurs are very commonly slightly lower than the maxima points of the corresponding sounds in health. Certain conditions of the aorta or pulmonary artery, of the lungs, pleura, or mediastinum, also throw murmurs into unnatural sites, by changing the position of the heart. Pleural fluid accumulations have more influence in changing the audible point of apex, than of basic, murmurs; because, while detruding the heart as a whole sidewards, these accumulations push the apex further away than the relatively fixed base.

728. The state of the heart's substance also may exercise a direct influence on the intensity of murmurs. Thus hypertrophous texture, lying, in the course of the circulation, behind a murmur, will intensify this, if it be direct; exercise little effect on it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant aortic murmur. Hypertrophous texture, lying in front of a murmur, is without effect upon this, if it be direct; intensifies it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant mitral murmur. Dilatation and enfeebling affections of the heart's substance weaken any direct murmurs through orifices on which such substance may play. Dilatation lessens the force of a regurgitant murmur, in so far as power of backward propulsion is concerned; but as, on the other hand, it allows of considerable accumulation of blood for regurgitation, the weakening influence is somewhat counterbalanced.

729. The physical conditions of a given murmur positively existing at a given orifice, the character of that murmur may be seriously affected by the anatomical state of other orifices. Thus, aortic constriction may be almost murmurless, if there be any great coexistent mitral regurgitation, the aortic current is so materially weakened by such regurgitation.

730. Of two murmurs, produced synchronously at different orifices, one may mask or cover the other. An aortic constrictive and a mitral regurgitant murmur may exercise this reciprocal influence; and a mitral regurgitant may completely drown a tricuspid regurgitant murmur. In the interspace between the maxima points of two synchronous murmurs, however, a spot may generally be detected where the intensity of murmur is less than at either, and the quality, as well as the pitch, different.

731. An hypertrophous and widened state of the aorta will intensify its own regurgitant murmur; and if slight pouching exist at the sinuses of Valsalva, the rippling direction thereby given to the current will intensify both a direct and a regurgitant murmur. The state of the blood, too, is important: spanæmia greatly strengthens the force of murmurs of all kinds. The pulmonary veins and superior cava may be the seat of spanæmic murmur,

simulating cardiac murmurs—especially systolic at the apex and diastolic at the base.

732. Again, modified conducting power of adjoining textures may lead to error: thus an aortic constrictive murmur may be heard better to the left than the right of the sternum, if the left lung be solid and the right emphysematous. Lastly, excessive irregularity and frequency of the heart's contractions, may make it impossible to determine the synchronism of a murmur.

733. The physical conditions of any orifice, or of its valve, giving rise to murmur, are liable to change—alteration in existing murmur will or will not occur, according to the nature of that change. First, the most common occurrence of the kind is aggravation of any given anatomical state, constrictive or regurgitant; such aggravation will, as a rule, exercise but little appreciable influence on the murmur, and certainly no influence which can during life be assigned with surety to its cause.

Secondly, cases are pretty frequent in which disease of constrictive effect existing at an orifice, the valves, becoming more gravely diseased by-and-by, permit reflux; or what is more rare, the conditions of regurgitation having taken the lead, those of obstruction follow. In either case a single gives place to a double murmur.

Thirdly, the conditions of murmur both of direct and of reflux mechanism, may be present at the same time in the same valve, but so related that now one, now the other, form of murmur is alone audible. Of this peculiar condition I have already spoken [696, *a*].

Fourthly, a yet more rare, and, as far as I know, undescribed, condition, is that in which a regurgitant murmur wholly disappears at an orifice, giving place to an obstructive one. I believe very confidently I have observed this at the mitral orifice; but I am without the evidence of *post-mortem* examination. The following facts demonstrate the reality of the occurrence at the aortic orifice.

December 7, 1860. Mr. . . . , ætatis 57; in good condition; sleeps badly, but with equal comfort on both sides; no dreams; can walk *five* miles an hour with ease; walked forty-two miles in nine hours and forty minutes a year ago; can walk up hill; complains specially of a disagreeable sensation at and below the left nipple, not amounting to actual pain, nor attended with faint feeling, and relieved by pressure; once felt a sensation, after a violent pedestrian effort, as if something had given way within the chest, and exclaimed, "Good God, I wish I had not done that." Never had rheumatic fever nor gout; pulse said to have been irregular from birth; certain that it was ascertained to be so at ætatis three; pulse now irregular, of medium frequency, not visible at radials, at one ulnar only, other arteries not looked at.

Heart examined in standing and sitting postures; action somewhat too diffused, and impulse too forcible; deep-seated percussion-dulness slightly in excess; medium-pitched, prolonged, blowing, diastolic, basic murmur, rather louder at the top of the sternum than at the precise base; slightly less resonant under percussion at the right than the left sterno-clavicular angle, also at the left than the right interscapular region—no pain in either of these situations. From these facts the diagnosis followed: moderate dilated hypertrophy of the heart; aortic regurgitation; calcification of the arch of the aorta [274]; and possibly some slight dilatation of the arch. I never saw the case, in life, again.

This patient died at the beginning of April, 1861. I then learned that he had, from time to time, been seen by Dr. Watson, in whose notes the existence of diastolic murmur at the base had been recorded as early as June, 1860. Dr. Watson saw him for the last time in February, 1861, and then to his surprise ascertained that of the diastolic murmur no trace impressed the ear, while a basic systolic murmur had now become loudly audible. The mystery, we found, received an easy solution in the state of the aortic valves. The substance of these valves had been considerably destroyed—to such extent as to admit of free reflux. But to the edges of the injured valves were attached masses of very soft fibrin, of such bulk as to block up the orifice of the vessel completely when looked at from above. Obviously this fibrinous plug, of relatively recent formation, must at once have prevented regurgitation and opposed a formidable barrier to the direct current from the ventricle. There was besides a recent perforation of the long tongue of the mitral valve, which may possibly, though to a considerable extent plugged with fibrin, have intensified systolic murmur. The arch of the aorta was slightly dilated, atheromatous but not calcified; the left heart slightly enlarged and hypertrophous.

734. It follows from this review that the diagnosis of valvular disease, though in the majority of cases sufficiently easy, must prove occasionally beset with various and serious difficulties—difficulties in certain instances so numerous and so singularly combined, that it would savor more of temerity, than of experienced judgment, to venture on a positive opinion. But such cases are of rare occurrence.

735. *Symptoms.*—The really important symptoms of each form of valvular disease are to be gathered, as matters of necessary inference, from the statements already made concerning the influence of each on the different capillary systems. There are a few functional effects which may be considered common, though by no means to precisely the same amounts, to all of the class.

Thus loss of strength and loss of ability for exertion, while relatively speaking the flesh is well maintained, prove very usual effects. Imperfect rest, startings from sleep with affright, and dreams of more or less distressing character, indicate the disturbed circulation through the nervous centres, entailed by the mechanical difficulties in the heart. As a rule, rarely broken, patients with valvular disease lie with the head high—the position of some in bed is rather that of sitting than of lying. But the rule is sometimes broken: I knew a case of enormous dilated hypertrophy with aortic constriction and regurgitation and mitral regurgitation, in which night-dyspnœa was greatly easiest when the head was kept on a slightly lower level than the shoulders.

Various paræsthesiæ may be felt in the cardiac region—or actual pain varying in degree between a slight aching sensation and the intensity of genuine angina. It has been plausibly enough suggested that atheromatous, and more particularly calcified disease of the valves and orifices, by interfering with the easy stretching and retraction of those parts, may explain the different varieties of painful sensation. I have thought, from what has fallen under my notice, that pain is more commonly connected with disease of the aortic, than of any other valves.

Palpitation, a frequent attendant on valvular disease, is in the main rather the offspring of the coexistent states of nutrition, capacity, and innervation of the heart, than of the mischief in the valves *per se*. At least it is certain any given form of valvular disease may reach any given degree of development without habitual palpitation proving a necessary effect.

Professor Seaton Reid has published a case showing that the peculiar form of disturbed respiratory rhythm, with passing apnœa, which had commonly been held to denote with surety fatty metamorphosis of the heart [663], may occur in connection with aortic and mitral regurgitation and dilated hypertrophy, though the sarcous structure be perfectly free from fatty change.[1]

736. *Mortality, Duration, and Prognosis.*—That the mortality due to valvular diseases in this country steadily maintains a high rate year after year, is as certain, as it is impossible, even approximately, to estimate the real amount to which they destroy life. The nomenclature adopted by the Register-General would, in itself, prove fatal to any attempt at roughly accumulating evidence concerning the mortality from these affections; but the statistics of the subject will, under any circumstances, always remain beset with inherent difficulties.

Nor, viewing all forms of valvular impediment as a single group, is it possible to make any statement of the least utility on the question of duration. Nay, even if any individual disease of the whole number be selected, the extremes of brief and of protracted course are found to lie so widely apart, that it becomes practically useless to strike a mean. Thus mitral regurgitation may destroy life—or at least act as the chief agent in destroying life—within a year from the first appearance of its physical signs; and yet signs as indubitable of mitral regurgitation have been known to exist in other cases for a lengthened term of years without the smallest subjective disturbance occurring. The amount of the valvular impediment, the diathetic state of the patient, and the condition of the heart's substance, sometimes afford a clue to this difference: but often the mystery proves inexplicable. Meanwhile we must be content to register the unexplained truth, that all forms of valvular disease may, on the one hand, prove the main instruments in rapidly killing, or, on the other, remain for a well-nigh indefinite period subjectively latent.

As matter of clinical experience, the chief valvular derangements may be placed in the following descending series on the basis of their relative gravity—that is, estimating this gravity not only by their ultimate lethal tendency, but by the amount of complicated miseries they inflict:—

Tricuspid regurgitation;
Mitral constriction and regurgitation;

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[1] Dublin Hospital Gazette, 1860.

Aortic regurgitation;
Pulmonary constriction;
Aortic constriction.

Of tricuspid constriction and pulmonary regurgitation too little is practically known to warrant us in assigning them their position in the series.

But there is a very important aspect in which the relative danger of these affections would place them in a widely different order—I mean that of their relationship to sudden, really sudden or instantaneous death. Taken as a group, valvular impediments cannot fairly be cited as frequent causes of sudden death; but there is one among the number of which the tendency to kill instantaneously is so strong, that the fact must always be borne in mind in estimating its prognosis—and that one is aortic regurgitation. The first example of the kind, which drew my attention to the subject, occurred in 1852;[1] since that time I have collected certainly eight, and probably eleven, similar cases. The manner of death is clearly syncopal; but the immediate mechanism, whether mechanical or dynamic, is difficult enough of comprehension. I have known death take place during the act of walking, of eating, of speaking —while the patient was emotionally excited, and, *per contra*, at a moment when he was perfectly calm. And a very singular proposition, which flows directly from my cases is, that the more pure and uncomplicated the regurgitation, the freer the heart from any other form of disease, the more likely is the individual to be cut off without a moment's warning. Thus, on the one hand, I have watched many a case of aortic regurgitation with mitral disease and aortic constriction, dilated hypertrophy of the left heart, and eventually tricuspid regurgitation, where death was slowly worked out through the sum-total of subjective and objective miseries of the latter form of valvular disease; while, on the other hand, in several of my instances of sudden death, not a single symptom existed to make the victim believe there was aught amiss within the heart. Here is a case in point. A man, aged about thirty-five, presented himself for insurance. He was a very picture of robust health, and had never had a symptom of disease connected with any organ in his body. Almost purely as matter of form I put the stethoscope to the chest; my attention was at once arrested by a loud murmur, which proved to be basic and diastolic. The arch of the aorta was sound; there was neither hypertrophy nor dilatation of the ventricle; the superficial pulses were markedly visible. To the astonishment of the proposed insurer himself, and to the passing chagrin of the Board, this, to outward seeming, "model life" was refused. The man dropped dead in the street within a fortnight of the refusal.

Sudden death may, then, I think, be regarded, if not as an apanage, at least as a likely enough contingency, of aortic regurgitation. Now sudden death, it is true, does in very rare instances occur in other forms of valvular disease, but then it is purely *accidental*, and is in nowise an attribute of the impediment *per se*.

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[1] I then simply noted the fact, without at the moment foreseeing its importance.

The condition of the heart's substance will help in forming our prognosis. Dilatation of the cavities always renders any valvular condition more dangerous: and as a rule, hypertrophy, though to a less degree, has the same kind of effect. Hypertrophy of the left ventricle, however, and directly as its purity, may mitigate the effects of aortic constriction; while in the case of regurgitation, its influence seems practically contradictory. On the one hand, it certainly increases the general and local evils of the reflux; and yet, on the other, mysteriously lessens the likelihood of sudden death.

In estimating the prognosis in any particular case, the amount of secondary mischief, pulmonary, systemic, renal or cerebral, affords valuable aid. The "physical signs proper" are always useless, sometimes deceptive, as prognostic guides. No greater error can be committed than that of supposing the danger of valvular diseases, as a class, may be estimated by the amount of murmur they habitually entail; *exempli gratiâ*, tricuspid regurgitation and mitral constriction, which hold so high places in the scale of fatality, are precisely the two with least constant murmur. This is sufficient proof that implicit faith—nay, any faith—in respect of prognosis, must not be put in the conditions of valvular murmurs; and it may be well here, again, to remind the younger reader of what may be fully gathered from the descriptions in the first part of this volume, that there is no direct connection between the amount and danger of disease at an orifice and the intensity of an existing murmur; the very *weakness of a murmur may, indeed, be a fatal sign.*

But the natural history of valvular disease is probably not one of wholly unmixed evil. There is an *à priori* probability, at least, in the view that, where the minute vessels of the brain are positively and gravely diseased—atheromatous, calcified, inelastic, weakened, and fragile—life may be prolonged through the interposition of a constricted state of the mouth of the aorta. So, too, it is conceivable the lungs (where the disposition to congestion and the reality of capillary weakness have pre-existed) may be saved from grave textural disease through the instrumentality of tricuspid regurgitation.

And, again, it is true, though to all seeming paradoxical, that the sufferer from certain forms of valvular disease, will be placed in a better position, especially as regards probable prolongation of life, by having one or more other forms of disease of the orifices added to that by which he is already afflicted, than if he retained his original impediment uncomplicated. Take the instance of a sufferer from grave mitral obstruction, who, on the eve of pulmonary apoplexy or œdema, becomes the subject of sequential tricuspid regurgitation, which, though profoundly mischievous in itself, nevertheless, *pro tempore*, wards off pressing danger by relieving engorgement of the lungs and transferring intra-vascular strain from the pulmonary to the systemic capillaries.

737. *Treatment.*—Whatever be the mechanical condition of diseased valves, and whatever be the nature of the diseased action—inflammatory, fibrin-depositing, fatty, calcifying—that has impaired their freedom of function, they lie themselves without the pale of direct treatment. Valvular disease once chronically established cannot be cured: we can neither remove deep-seated induration-matter, atheroma, nor calcareous substance, nor lengthen tendinous cords that have been shortened by morbid processes. Hence the importance of at once guarding as much as possible against one of the frequent causes of acute endocarditis—namely, rheumatic fever, and of preparing for the decisive treatment of that inflammation the moment there is reason to suspect its immediate advent. Of the prophylaxis of non-inflammatory valvular changes, nothing can at the present day be said.

738. But though these diseases be anatomically incurable, their worst functional effects may be long, in some cases indefinitely, averted, by measures accordant with a common-sense view of their nature and ratified by experience. Whatever be the valve implicated, the treatment is directed not towards its own disease, but towards the moderation or prevention of hypertrophy of the muscular substance of the heart or dilatation of the cavities—the control of extra strength or the support of undue weakness—and the removal or relief of any symptoms that may arise. Practically the treatment of valvular diseases comes to be that of hypertrophy or dilatation, due regard being had to the influence exercised by the mechanical obstruction on the character and tendencies of those conditions of the heart's substance. Thus, if with valvular disease there coexist dilated hypertrophy, occasional very moderate venesection, or the abstraction of blood by cupping or leeching over the præcordial region, will be advisable: more so in cases of superadded mitral regurgitation than of aortic constriction, for reasons easily referable from the influence of such hypertrophy in these two valvular diseases. Profuse bloodletting is, under all circumstances, absolutely improper: it cannot remove either the disease of the valves or of the heart's substance, and may induce anæmia, excitement of the heart, and early dropsy. If dilatation exist, with or without attenuation, venesection is decidedly contra-indicated; three or four leeches may, however, even under these circumstances, be applied to the cardiac region for the relief of palpitation, anginal feelings, or sudden engorgement of the right cavities. Dry-cupping and mustard poultices should, however, first be tried: they often give quite as much relief, and without any concomitant sacrifice.

Of the value of *diuretics*, when dropsy has appeared, no doubt can be entertained: but long before matters have proceeded to this point, medicines of the class seem useful, by diminishing the quantity, without impoverishing the quality, of the circulating fluid, and so lessening the tendency to clogging of the intra-cardiac circulation and the proneness to palpitation. The acetate, nitrate, iodide, and bitartrate of potass (the latter in two-drachm doses largely diluted), nitric ether, compound tincture of iodine, the infusion and spirits of juniper, the decoction of chimaphila, and other agents of the class may be variously combined and successively employed. The action of these diuretics is facilitated by occasional small doses, at bed-time, of blue pill and squill. When the kidneys are much congested, removal of that congestion must be effected, wholly or partially, before the organs will act. Cupping to a small extent or dry-cupping in the renal regions sometimes exercises a remarkable effect in facilitating the action of diuretics by removing congestion: in the same way is to be explained the favourable influence of mustard poultices and even *blisters* to the loins. Urine, more or less impregnated with albumen, previous to these measures, sometimes becomes perfectly free, to ordinary tests, of that principle after their employment.

Regularity of alvine discharge, in all diseases important, is particularly essential in these cases, to prevent engorgement of the liver, and obviate the necessity for effort in the act of defecation. Continued purgation is, however, to be avoided prior to the occurrence of dropsy.

If at any time the stomach be loaded, and its replete state excite palpitation, an *emetic* seems clearly indicated: the sulphate of zinc is the fittest. But where the circulation is very much embarrassed, emetics sometimes increase considerably that embarrassment, and unhappily it is far from easy to fix beforehand the probable influence of an emetic in any particular case. If there be doubt, it is better to refrain, and allow the stomach to free itself gradually in the natural manner. Dr. Hope was of opinion, and with much *à priori* reason in his favor, that a state of prolonged nausea, by causing languor of the circulation, promoted the formation of fibrinous coagula within the heart.

Where the lungs tend to engorgement, especially in mitral disease, *expectorants*, either ipecacuanha, lobelia, stramonium, or squill and ammoniacum or senega, according to the active or passive character of the symptoms, become necessary. Dry cupping of the chest, sinapisms, and blisters materially relieve such engorgement. *Antispasmodics*, chloric ether, ammonia, &c., are necessary in paroxysms of dyspnœa. *Opiates* cannot be dispensed with at night in advanced cases; but where the valvular obstruction is considerable and the heart weak, caution is required in their exhibition. *Antacids*, carminatives, and light bitters, and other stomachic medicines, relieve the gastric discomfort so common in the victims of valvular disease; but the latter must not be permitted to an extent to stimulate the appetite to any great amount.

The propriety of administering mineral *tonics* will, in the main, turn on the condition of the heart's substance—whether dilated or hypertrophous—a subject already considered. Theoretically, invigorating tonics are more advisable in aortic constriction than in mitral regurgitation. Anæmia peremptorily calls for iron.

Issues or *setons* to the præcordial region sometimes relieve local pain and discomfort; they are otherwise valueless. At the very earliest period of chronic inflammatory changes in the valves, it is possible that iodduretted frictions may promote absorptive action.

The treatment of dropsy has already been described [619].

In regard of diet, no constantly applicable rules can be laid down, except that moderation is important both in solids and, especially, in fluids. Exercise should never be pushed to fatigue, and laborious efforts of all kinds systematically avoided. When the dyspnœa is of serious character, the toil of mounting a flight or two of stairs, especially if the patient be obese, is more than he dares encounter: his instinct apprises him of the imminent risk of the attempt. Under these circumstances the common plan is to recommend the patient to occupy the ground floor—a mode of living having its own hygienic disadvantages, unless in houses constructed on an unusually large and commodious scale. Mr. W. B. Hutchinson has, in a case of this kind, succeeded in enabling the sufferer to use a sleeping-room at the top of the house, much to the improvement of his general health, by having him raised in a chair suspended by solid pulleys to the ceiling of the staircase—something on the plan of the well-known contrivance at the Coliseum, in the Regent's Park.

The applicability of digitalis in the management of valvular diseases is, I think, more limited than is commonly conceived. I presume that in regard of motor-sedative influence, its efficacy is to be measured not by altered force, but by altered frequency, of beat. Now, theoretically, if the beat be notably slackened, where embarrassment of the intracardiac circulation already exists in consequence of valvular difficulty, that embarrassment must be increased. And, as matter of experience, there can, I think, be no doubt that evil of this form is occasionally produced by the hap-hazard administration of digitalis—actually eventuating sometimes in coagulation within the cavities. It is true various cardiac paræsthesiæ, and even downright pain, are occasionally relieved in so remarkable a manner by the drug, as to thus justify its well-known epithet—"the opiate of the heart." But it seems a mistake to purchase the relief of uneasiness and pain at the risk of shortening existence by yet further increasing the difficulty of circulation through the organ.

Something will depend on the nature of the existent valvular difficulty. Digitalis may be a safe remedy enough, where a not over-constricted aortic orifice is acted on by a markedly thickened left ventricle; of its danger in cases of serious mitral and tricuspid difficulty I entertain no doubt; while in aortic regurgitation, with its specific leaning towards sudden death, its influence cannot be otherwise than hazardous.

If it be decided to employ digitalis, the infusion is probably its safest form—the diuretic action, commonly induced, contributing to ward off ill effects. Of digitaline my experience is limited—the chief value of the alkaloid seems to consist in the accuracy with which the amount of really effective principle may be dosed. I confess I have failed to observe the remarkably beneficial effects from its administration that appear to have impressed some persons.

§ IX.—ANEURISM OF THE HEART AND VALVES.

I.—Heart.

739. Aneurism of the heart occurs in two forms, corresponding to the fusiform dilated aneurism, and the lateral simple sacculated aneurism, of arteries (*vide* Diagram, figs. 1, 3, [824]): that is, a general and tolerably equable dilatation of a portion of the wall of a ventricle exists, or a pouched fulness rises abruptly, with or without constricted orifice, from the ventricle. Of either kind, aneurism is almost peculiar to the left ventricle; of seventy-four cases, collected by Mr. Thurnam, fifty-eight were examples of the disease in the left ventricle—while in no single instance was the right affected. The compound sacculated aneurism of arteries, with injury to their walls (Diagram, fig. 4, [824]), is also imitated by the heart, when destruction of the endocardium and, more or less extensively, of the nearest strata of muscular fibres, precedes the pouching process.

Sacs of recent formation are generally provided with a wide orifice; as the sac ages, the opening relatively loses in diameter, while its lips grow prominent, well-defined, and fibroid.

There may be little or no bulging on the pericardial surface of the ventricle, indicating the seat of the disease; or an elevated prominence, varying in size from that of a nut to that of well-nigh the heart itself, may exist. And when, as is not very uncommon, the septum is the seat of the aneurism, the sac need not in the least alter the external form of the heart.[1]

Aneurismal sacs have been found empty, or have contained coagula, either laminated or amorphous—all three states with about equal frequency. In the vast majority of cases a single sac only has existed; but not a few examples of two, and a rare one of three or even four coexisting sacs, are to be found recorded in periodical works. When numerous, the sacs may coalesce.

The reality of processes of curative tendency occurring in cardiac aneurism has now and then been substantiated; indeed, actual cure has occasionally been accomplished by the complete obliteration of the sac with fibrin.

740. In the majority of cases aneurism forms slowly, dependent,

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[1] Univ. Coll. Museum, No. 4592; sac protruding through septum below aortic valves, communicating with the left, bulging into the right, ventricle.

as it is, for existence on chronic changes in the substance of the heart; in some cases, however, its formation is an acute process, induced by ulceration or rupture of the endocardium and contiguous fibres. Once produced, the structure forming the wall of the sac may undergo secondary changes in the shape of cirrhotic, fibroid, cartilaginiform, or ossiform infiltration.

741. Greatly more frequent (as 3 to 1) in males than females—hitherto most commonly observed between the ages of twenty and thirty and in very advanced life, though few ages are actually exempt from the possibility of its occurrence—having the groundwork laid for its formation in inflammation, fatty change, simple softening, cirrhotic [686], or pseudo-fibrous infiltration of the heart's substance—aneurism has been immediately traced, in a certain number of instances, to external injury, violent efforts, forced retention of the breath, and similar agencies, suddenly throwing an intense strain on the walls of the left ventricle.[1] In the majority of cases its clinical, as well as its anatomical, origin is slow and insidious—in fact, latent.

742. *Symptoms.*—Under all circumstances, whether the production of the aneurismal pouching be an abrupt and rapid, or a deliberate and slow process, the symptoms are obscure.

(*a.*) Where the onset has been presumedly sudden, the heart's texture lending itself through local structural weakness to quasi-instantaneous bulging, the occurrence has beyond doubt been attended with a certain number of grave cardiac disturbances—such as severe præcordial pain, orthopnœa, alarm and general agitation, dread of dissolution, syncopal tendency and frequent small, irregular, languid pulse. But obviously there is nothing distinctive in these symptoms: they indicate that the heart has received a deep shock of some kind, and nothing more.

(*b.*) On the other hand, where the disease is of slow origin and course, its symptoms are non-specific, and do not seem to become serious, unless itself is carried to a great amount, or dilatation, with more or less hypertrophy, is added. The effects of such dilatation are then developed—systemic stagnation and its attendant evils in more or less complete array. At all events, it will be admitted that past records do not supply the materials of a clinical history of aneurism, clearly distinct from that of affections of the ventricles and of the orifices, however complete an anatomical one they have been made to yield by the zeal of Mr. Thurnam. Every known symptom of cardiac disease has been present, it is true, in these cases; but, as there either were positively, or may possibly have been, other morbid states present (I refer to narratives which make no affirmation as to the absence of such states), capable of causing the symptoms in question, it would be perfectly unjustifiable to ascribe them to aneurism.

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[1] Prolonged stage-effort has been assigned as the cause of the cardiac aneurism of the great French tragedian, Talma; but as he had long labored under stricture of the rectum, it appears to me the perpetual strain in defecation must have played the part of a much more active cause.

743. *Physical signs.*—And there is as great a dearth of knowledge of positive physical signs. The position of the heart's maximum impulse may perhaps be transferred from the apex to the base, where a sacculated aneurism springs from the neighborhood of the base of the ventricle; probably, if the sac were prominent, the action of the heart would be attended with pericardial rub, not only systolic, but diastolic. Actual tendency to present externally, after adhesion of the sac to the chest-wall, has been observed; the peculiarity does not appear to have led to successful diagnosis—though it is conceivable the nature of the affection might at least be surmised through its existence. Systolic murmur of blowing quality, of maximum force at the left apex, has been observed, undistinguishable from the murmur of mitral regurgitation. Though seemingly probable, *à priori*, that a double murmur might be produced by the ingress and egress of blood from the sac, where the orifice of this was narrow, experience shows that the diastolic portion may be completely wanting.[1] The signs of dilated hypertrophy frequently, of valvular disease sometimes, are coincidently met with.

744. Death may occur suddenly from rupture of the sac into the pericardium, or, through an adherent pericardium, into the pleura: in the majority of instances the patient is slowly worn out with the symptoms of dilatation.

745. There is no special treatment for this affection: the symptoms and signs guide the physician to the adoption of the measures best adapted for hypertrophy or dilatation: the effects of systemic stagnation are to be averted or removed by the plans already described.

II.—VALVES.

746. Aneurism, pouching, or sacculation has been observed in the mitral, tricuspid and aortic valves—habitually, as might be expected, with the concavity of the sac bearing in the direction of regurgitation. Doubtless the local distension is produced by the force of the falling column of blood acting on textures weakened in resistant power by chronic impairment of nutrition. Generally very small, the sac may equal a hazel-nut in size; it may undergo perforation.

In a specimen of sacculation of the long tongue of the mitral valve now before me,[2] the sac bulges, contrary to the rule, in the direction of the ventricle. It might lodge a small pea in its interior; and is much thinned, well nigh perforated, at its apex. It is difficult enough to understand how undue strain could be exercised on the mitral valve in this direction; and the mechanism of aneurism thus occurring, as it were, with the blood-stream, is obscure enough.

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[1] *E. g.* In Case V. of Dr. H. Douglas's Collection of Cases of Heart-disease, Edin. Monthly Journal, 1850.

[2] Univ. Coll. Museum, No. 4166.

The clinical history of valvular aneurism is as yet a blank.

§ X.—RUPTURE AND PERFORATION OF THE HEART.

I.—COMPLETE RUPTURE.

747. Rupture of the heart's substance into the pericardium, from all causes, spontaneous and other, indiscriminately, takes place with much greater frequency in the left ventricle than any other part of the organ:[1] but, if cases originating in external violence alone be considered, the right ventricle has, according to existing records, suffered more frequently than the left, in the proportion, according to Ollivier, of eight to three.

748. Variable in size and form, sometimes smooth, sometimes ragged at the edges, the inner and outer openings—that is, the pericardial and the endocardial—may correspond or not; in the latter case, a sort of sinus exists in the wall of the ventricle, connecting the two. There is usually a single rupture only, but so many as five have been seen; sometimes a single opening on the inner surface of the ventricle communicates with two or more on its pericardial aspect. The strata of muscular tissue next the endocardium, again, may be pretty extensively destroyed, while a tiny perforation only in the pericardium can be discovered. Although there is every reason to believe that, in the great majority of cases, rupture of the heart is perfectly sudden in its occurrence, it is clearly a gradual process in some rare cases; for instance, where hemorrhagic softening of tissue has led to its occurrence: under all circumstances, the final breakage of the last few muscular fibres and outer serous membrane, that separate the pericardial and endocardial cavities, is instantaneous. The fissure runs very nearly three times as often parallel to, as at right angles with, the main fasciculi of the heart's fibres.

749. Considerably more frequent in males than females, in the ratio of 36 to 16, rupture of the heart is favored by advanced age: it becomes comparatively frequent after the fiftieth, still more so after the sixtieth year. Immediately induced by effort of some kind, by fits of passion, by great and abrupt thoracic congestion, as though sudden immersion in the cold bath, by blows and other injuries to the præcordial region, the way is paved for its occurrence by various textural changes of the heart's substance or aorta: as fatty accumulation under the pericardium, combined with muscular softness; intra-sarcolemmous fatty change; softening of undetermined kinds; dilatation with attenuation; local suppuration, ulceration, possibly gangrene; hypertrophy, with, probably, fatty change superadded;

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[1] In 52 instances of rupture collected by Gluge (Path. Anat.) the left ventricle was the seat of the rupture in 37 cases; the right ventricle in 8; both ventricles in 2; the right auricle in 2; the left auricle in 3.

hydatids; calcification of the ruptured tissue; aneurism of the left ventricle; local destruction of the endocardium; apoplexy of the heart (the question has, however, been raised, whether in cases where blood-infiltration and rupture have been found together, the former may not have depended on gradual advance of the latter); coarctation of the arch of the aorta; and, as in the case of George I., aneurism of that vessel. In all probability, though old narratives say the contrary, the heart's texture is never perfectly sound; even where the cause of the rupture has been external violence, some alteration of tissue has been generally found by recent observers.

750. *Symptoms.*—In the majority of cases, rupture of the heart, if actually complete, at all extensive, and instantaneous, kills instantaneously. The hand is suddenly carried to the front of the chest; a piercing shriek uttered; some convulsive twitches occur, and the patient expires: or sudden loss of consciousness, from which recovery never takes place, marks the event.

751. There is a second class of cases, in which, from the plugging of the fissure by coagula, the amount of extravasation of blood into the pericardium is insufficient to stop the heart's action at once. Under these circumstances, a patient has beeen known to survive fourteen hours, with pallor, cardiac anguish, clammy sweats, coldness of the surface, feeble fluttering pulse, and sighing respiration; eventually going off quietly in a state of coma. The fact of death occurring so slowly suggests the question whether the permanent closure of a fissured opening does not fall within the range of the possible; and certainly one case of sudden death from rupture has been recorded, in which a former rupture was discovered, firmly filled by a fibrinous coagulum adherent to the wall of the heart. I have before me a preparation, in which an attempt at plastic closure is shown. Towards the apex of the right ventricle lies a fissure about five-eighths of an inch long; at the upper angle appears a minute aperture leading into the ventricle; while the remainder is filled with lymph, adventitious (?) muscular fibre, and wavy fibres with round particles and granules in their interstices (Univ. Coll. Mus., No. 4077). The clinical conditions observed at the close of life in this case may advantageously be placed side by side with this brief notice of the state of the heart.

William Hutchinson, ætat. 48, presenting all the signs and symptoms of general dilatation, with hypertrophy, of the heart (the organ weighed twenty-two ounces), mitral and tricuspid regurgitation, enlarged liver, pulmonary apoplexy and anasarca, had slightly improved in the majority of his symptoms during three weeks' stay in hospital, when a change for the worse, attracting the immediate attention of the nurses, occurred on the morning of the 25th August: the countenance had become of pale yellowish earthy aspect; the conjunctivæ bloodless; the face and thorax covered with cold sweat; the respiration labored and frequent; the pulse, weaker and smaller than previously, uncountable from frequency; feet considerably œdematous and cold; no pain complained of; restlessness intense—change of posture almost every minute; tongue particularly clean; bowels naturally open. During the night of the 25th, insomnia, constant restlessness; dressed himself and sat on side of bed greater part of the night wishing to go home. August 26th. In the morning: does not know his children; body exhales an earthy, and somewhat putrescent odor; extremities cold; pulse almost imperceptible, uncountable from frequency; respiration more difficult and frequent than yesterday; cold sweats: so late as 3.30 P. M. this day he got out of bed to sit up; passed urine under him; at 5.0 P. M. hacking cough set in; shortly after, the respiration became infrequent, occurring at such rare intervals, that each breath seemed to be his last. . . . Sank at 5.59 P. M., without any change of symptom. On examination (made, in my absence, by Dr. Hare) about two ounces of dark blood, partly coagulated, partly fluid, were found on the surface of the heart and root of the aorta; after careful search, a minute aperture, barely admitting an ordinary sized pin, was detected close to the apex of the right ventricle, and traced to communicate with the interior of that cavity in an oblique direction downwards, and to the left. (U. C. H., Males, vol. viii. pp. 334—354.)

The two conditions of the blood in the pericardium (coagulated and fluid) point to two distinct acts of hemorrhage; the one coincident with the first and nearly closed rupture, the other with the final perforation. The probability is, the first rupture was effected during the night of the 24th, or morning of the 25th; and that consequently the patient survived the occurrence thirty hours on the lowest computation.

752. A third class of cases exists, in which sudden cardiac anguish, attended with a sense of constriction, extreme dyspnœa, pain extending from the præcordial region to the left shoulder, coldness of the surface, giddiness and faintness, cramps, and small irregular pulse, are observed—the whole series undergoing remission, nay, actually, it is said, totally disappearing temporarily, again to return with greater intensity than before, and close in death. In instances of this kind, in all probability, the heart's fibres have given way in successive layers.

753. *Treatment.*—The greater number of cases of at all extensive rupture have terminated fatally, before medical attendance has been procured. Should the patient be still living, when first seen, such treatment should be employed as a common-sense view of the symptoms would suggest; for it is more than probable that the precise nature of what has occurred will rarely be diagnosticated with surety. Theoretically, the clear indication is to maintain the circulation in movement, with as little work on the part of the heart as possible; the head should be placed low, sedatives and slight stimulants administered, the surface kept artificially warm, and the very slightest movement of the body, as far as possible, prevented. If reaction occurred, blood should be cautiously taken from the arm.

In instances of slow perforation, of which an illustration has been given, the nature of the occurrence is, in the first place, beyond the reach of sure diagnosis. In an individual, dying more or less rapidly of profound organic disease of the heart, the increased uneasiness and increased disturbance of the pulse, depending on perforation, might be taken for indications of polypoid formation within the cavities, or rupture of a portion of valve or chorda tendinea. Fortunately, however, the treatment will not err in consequence: in any one of these cases the immediate management is that fitted for nervous shock and cardiac obstruction.

II.—PARTIAL RUPTURE.

754. Partial ruptures of the heart, or of its valvular apparatuses, are not extremely unfrequent. Rupture of the papillary muscles, permitting the ruptured ends to float, as it were, free in the cavity of the ventricle; rupture of the substance of the mitral or tricuspid valves, or of their tendinous cords; and of the pulmonary and aortic valves, have all been observed *post mortem*, and their symptoms occasionally noted with more or less precision during life.

755. The immediate symptoms of the rupture of a chorda tendinea, especially if endocarditis have caused the accident, are not very distinctive. The difficulty of the circulation through the heart, however, is increased; the pulse becomes very irregular, and symptoms, precisely like those of the sudden formation of blood-concretions in the ventricles, make their appearance. The murmurs, obstructive and regurgitant, of the implicated valve ensue; but they may have pre-existed, as consequences of the endocarditis itself, and in that case positive diagnosis is an impossibility.

Sudden rupture of any number of chordæ tendineæ, or of a papillary muscle, when, *per contra*, occurring independently of acute disease of the heart, produces very definite symptoms. Cardiac pain and anguish; palpitation irregular in force and rhythm; small, irregular, frequent pulse; syncopal tendency, overwhelming dyspnœa, dread of dissolution, pallor, coldness of surface, jactitation of the limbs, all this suddenly occurring in connection with a regurgitant systolic apex-murmur, which had been known not to have existed previously, might guide to the diagnosis, but not justify a positive assertion on the subject.

In a case of rupture of one of the aortic valves, observed by myself, symptoms such as those just described were attended with regurgitant, without obstructive, basic murmur.

756. The character of the symptoms points to the propriety of administering stimulants and sedatives; sinapisms or turpentine fomentations to the cardiac region relieve the anguish and constriction felt in that situation.

§ XI.—MALPOSITIONS OF THE HEART.

757. Malpositions of the heart are congenital or acquired.

758. (A.) Of the *congenital* class the extra-thoracic, cephalic, and abdominal ectopiæ are totally without clinical interest. Not so, congenital malposition of the heart in the right half of the thorax; for this being a kind of displacement producible at all periods of extra-uterine life by a variety of diseases, it becomes of importance to have a means of positively distinguishing the malposition of

congenital origin. Now this means is furnished by the position of the liver and spleen; for, except in very rare instances, when the heart has formed and grown on the wrong side of the spine, the abdominal viscera, also, have been transposed. Where this guide is wanting, the distinction may be very difficult; and the observer is only justified in pronouncing the malposition of the heart to be congenital after he has succeeded in excluding every possible morbid source of acquired displacement.

759. (B.) The diagnosis of *acquired* displacements of the heart is to be made by the position of the apex-beat, and of the impulse of the organ generally, by the altered locality of cardiac percussion-dulness, and by the comparative intensity of the heart's sounds, more especially of the first, at different parts of the chest.

760. The varieties of acquired displacement, which possess practical importance, are three—namely, sideward detrusion and traction, elevation and procidentia. To very slight amounts all three are of very frequent occurrence, a necessary result of the looseness of the anatomical connections of the organ [1]. But, even when carried to extremes, these displacements produce little or no subjective disturbance; their clinical interest really depends upon the light thrown by their discovery on the diagnosis of various intra-thoracic diseases.

761. *Detrusion* may occur towards either side, but is more striking, and hence more readily discovered, in its minor degrees, when occurring towards the right than the left. In the former position the main impulse may be carried nearly as far outwards as the right nipple, between the fifth and seventh ribs. In the latter, the axillary region may pulsate markedly, while movement is scarcely perceptible, or actually null, at the true cardiac region; the apex is commonly raised by the width of an intercostal space above the normal level.

In both cases the base is less displaced than the apex; the organ not only changing its place as a single mass, but undergoing a rotatory movement, of which the base-region is the centre. Hence it is that the maximum point of the first sound suffers very much greater change of place than that of the second. It is noteworthy enough that the amount of displacement actually found after death is habitually less than the state of the physical signs during life has led the observer to anticipate. I have repeatedly noticed this where all necessary precautions (ligature of the trachea, fixing the heart *in situ* by long needles, &c.) had been taken to prevent the positions of the viscera from being notably altered by opening the thoracic and abdominal cavities.

By far the most frequent cause of lateral displacement of the heart is pleuritic effusion: above all, this is the affection which, in actual practice, most commonly carries the organ seriously out of its place. Perforative pneumothorax, with inflammatory liquid effusion, will push the displacement to its extreme possible limit.

Simple pneumothorax is so rare an affection in the idiopathic form, and, where perforative, is so rapidly followed by other morbid conditions of the parts concerned, that its detruding influence on the heart is rather matter of theory than of actual observation. Hæmothorax is rarely copious enough for the purpose; but in a case of aneurism of the arch, terminating by sudden death from extravasation of about two quarts of blood into the left pleural sac, the heart was found, *post mortem*, lying to the right of the middle line.[1] True hydrothorax being generally double, and consequently exercising about equal pressure on both lateral aspects of the heart, does not displace the organ sidewards. Intrathoracic tumors and aneurisms,[2] variously placed, occasionally push the heart to the right or to the left. Hypertrophy and emphysema, if limited to, or in great excess, in one lung, drive the organ proportionally to the other side. Pneumonic consolidation, especially on the right side, displaces the heart slightly in the opposite direction, provided the hepatization be on a massive scale, and especially if it be rapidly effected; but if the lateral displacement be at all considerable—if it equal an inch and a half—the presence of fluid in the pleural sac may, according to my experience, be looked upon as certain. Finally, hernia of the intestines through the diaphragm may be quoted as an infinitely rare cause of this variety of malposition.

The heart may be pushed forwards by emphysema; the displacement is with difficulty ascertained, on account of the presence of the thick stratum of distended lung in front. Intrathoracic tumors and aneurisms lying behind the heart push it forwards, the signs in the latter case especially are very peculiar; the simulation of cardiac hypertrophy may be very perfect.

762. *Traction.*—The heart may be drawn, as well as pushed, sidewards from its normal site. In cases of rapid absorption of right pleuritic effusion, the heart is occasionally dragged into the right thorax at the period of absorption, especially if that absorption be very rapidly effected. So, too, it may be displaced by an analogous mechanism in cases of unusually marked diminution of the bulk of the lung, induced by interstitial contraction of pneumonic exudation-matter. This is much better seen, however, in genuine pulmonary cirrhosis. Slight displacement of the sort occurs in simple (but not in emphysematous) atrophy, limited to one lung. I have seen the heart beating in the right thorax, where the sole explanation was to be found in great reduction of bulk of the right lung, through the contraction attendant on tuberculous infiltration, and on lessening area of large excavations.

763. *Elevation* of the heart above its natural level may occur in all varieties of enlargement of the abdominal viscera, and in cases of liquid and gaseous accumulation within the abdomen. The

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[1] Bell, U. C. H., Males, vol. vii. p. 169.

[2] To a very remarkable amount in Moriarty, U. C. H., Males, vol. xii. p. 5.

splenic enlargement of leucocythemia may permanently raise the organ an entire interspace and upwards;[1] enlargement of the liver from abscess will also raise the organ somewhat—a fact which, as I have elsewhere shown, may become very valuable in a case where the diagnosis between hepatic abscess and abscess in the abdominal wall over the liver chances to present unusual difficulties.[2] The heart may be very notably raised above the normal level by the contraction of a tuberculous cavity in the upper part of the left lung; or, to a less degree, through the diminished bulk of the apex which attends the earlier consolidating and atrophous stages of the disease.

As far as I have seen, ovarian dropsy is the affection in which elevation of the heart reaches its maximum. I have known it impossible to feel distinct impulse lower than the second interspace, though slight fluttering movement might be seen in the third.

The heart's apex may be carried upwards and outwards, while the base remains, practically speaking, fixed, in pericarditic effusion. Of this condition enough has already been said [54].

764. In *procidentia* the heart lies lower than natural, at the same time it generally sways a little to the right. In cases of prolonged debility, especially of adynamic pyrexia, there seems reason to believe that elongation of the great vessels occurs to a slight extent, and entails a proportional fall of the heart. In hypertrophy and dilatation, though the extension of the heart's outline downwards mainly depends on the increased bulk of the organ, it seems, especially in the former affection, in some measure traceable to lowering of level dependent on increase of weight. Mediastinal tumors and aneurisms occasionally push the organ more or less notably downwards. But it is as an attendant on emphysema of both lungs that this form of displacement is most frequent, and of most practical significance; in point of fact, such change of position of the heart is one of the most valuable signs of that pulmonary affection. Similar displacement may be caused by double bronchitis, but the acute inflammation rarely suffices to disturb the position of the organ, unless old-standing emphysema have pre-existed. Double hydrothorax does not push the heart downwards as invariably as might be theoretically expected.

765. There is no direct treatment of displacement of the heart. The organ is only to be restored to its place by the cure of the diseased states that have removed it therefrom.

§ XII.—INTRACARDIAC BLOOD-CONCRETIONS.

766. Coagulation of blood within the heart may occur either after death, or *in actu mortis*, or during life, and at various periods

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[1] Dujardin, U. C. H., Males, vol. v. p. 192, June, 1850; C. Hayes, U. C. F., Males, vol. x. p. 212; also Mr. ——, seen with Mr. Rayner, of Kingsland.

[2] Case of Fairbanks, Clin. Lec., Lancet, *loc cit.*, 1849.

more or less distant from its actual close. The characters of the resulting coagula are for the most part well-defined, and clearly significant of the date of their formation, whether *post mortem*, *intra mortem*, or *ante mortem*. But it must be confessed that instances, on the whole exceptional, every now and then occur, wherein it appears impossible to fix with certainty the period at which the coagulation-process really set in.

767. (A.) *Post-mortem variety.*—To this variety belong masses of variable size, consisting commonly of a stratum of loose, gelatinous and watery-looking, pale straw-colored, semi-transparent fibrin, coating, or not, grumous currant jelly-like coagulum of variable consistence. Structureless, non-laminated, interlaced or not among the musculi pectinati and columnæ carneæ, but firmly adherent to the endocardial surface, free from even the earliest indications of an attempt at vascularization, these concretions, more common on the right than the left side of the heart, may be grasped by and moulded to the surface of the infundibulum of the right ventricle, the pulmonary artery, and both its main divisions, and bear upon its surface a perfect mould of the pulmonary valves.[1]

Or, if at the period of death the blood have been notably deficient in fibrin, or if its fibrin have been of non-plastic quality, or if the manner of death have been calculated to obstruct coagulation, dark grumous currant jelly-looking masses may alone be found—distinct separation of the fibrin failing to occur.

768. (B.) *Variety formed shortly before and in the act of death.*—Fibrinous coagula formed shortly before death are non-watery, firm, opaque, laminated, and fibrillated, or even granular or lumpy, non-uniform in tint, being in some parts pale fleshy-looking; in others yellowish, or slightly greenish, marked on the surface with blood spots or even quasi-vascular looking streaks, adherent to the endocardium, and becoming rough and shreddy in the process of separation therefrom. They may be of uniform consistence throughout their mass; or if the diathetic conditions of the individual, the manner of death, or the atmospheric state be favorable to rapid physical and chemical change, the fibrin may soften, even become fluid, centrally; and under the latter circumstances bear the most deceptive naked-eye resemblance to pus.[2]

Now, precisely in proportion to the degree of perfection of these characters will the certainty arise, that the coagula presenting them were really found before death; and the conviction will be strengthened, if they are found on both sides of the heart. But on the other hand, I am persuaded coagula having all the characters of the first variety (A), are sometimes produced immediately prior to and during the final struggle.

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[1] U. C. H. Museum, No. 3636.

[2] *Vide* art. Adventitious Products, Cyc. of Anat. and Physiology, p. 114, 1848; here will be found the distinctive marks between genuine pus and softened fibrin. It seems of late years to be forgotten that the distinction in question is as old as the day of Dupuytren.

769. (C.) *Variety formed at a more or less distant period before death.*—The appearances of this variety vary considerably. Sometimes the concretion consists of very firm stratified fibrin, more or less markedly vascularized, and so firmly adherent that it can only be removed by the scalpel—the endocardium invariably undergoing injury in the process.

Or, again, the concretion occurs in the form of masses of various shapes, globular, elongated, ovoid, sessile, or pedunculated; ranging in size from a pin's head to a walnut and upwards; in color whitish, pale dirty straw, greenish yellow red, or reddish yellow; in structure obscurely fibrous, laminated, granular, and lumpy or amorphous; invested with a membranous coating identical in physical attributes and continuous with the adjacent endocardium. This variety of concretion is not vascularized; it has been supposed to undergo nutrition-changes through imbibition from the surrounding blood. Saline precipitation, and even a rude imitation of bone-formation, may occur interstitially within its substance.

The variety just described is solid, and of more or less closely uniform consistence throughout. But there is another form in which, while the globular outline and mode of endocardial attachment are identical, the concretion is hollow and sac-like. The contents of the sac are fluid, like water and blood mixed, or lees of wine, or of yellowish-green hue and puriform look. Sometimes the interior cavity is subdivided, giving a multilocular aspect. The cause of the sacculated appearance is simply the softening of the central portion of the original solid fibrinous mass—the coloring matter of the blood, associated in different amounts, giving the various tints to the fluid. The wall is often very thin, and its actual rupture, with effusion of the contained fluid, has in rare instances been observed. These saccular concretions may occur in any of the heart's cavities; they are said to form most frequently in the left ventricle—it has occurred to me to observe them oftenest in the auricles, attached to the appendices. They are, absolutely speaking, rare in all situations. They may be attached to the general tract of the endocardium or to its valvular portions. In the latter case, frequently pedunculated, their varying relationship to the connected orifice may give rise to very peculiar physical signs [696, *a*].

770. (D.) Lastly, fibrin may also be deposited from the blood at various, more or less lengthened, periods before death, in the form of small wart-like masses, or so-called *vegetations*. The most frequent seat of these bodies is on the surface and at the edges of the aortic and mitral valves; but, though exceedingly rare at the right side of the heart, there is no part of the endocardial surface on which they are not occasionally seen. Smooth or jagged, rounded or amorphous, they adhere either to an apparently unbroken endo-

cardial surface, or to fissures in the membrane, or amid the columnæ carneæ and the cordæ tendineæ.

Once deposited from the blood, the fibrin undergoes inspissation and hardening in the majority of instances—though the clinical history of cases sometimes seems to prove, they may maintain an unchanged condition of softness for years. It does so in this way: a given murmur has existed for years—the patient dies of an independent affection, and to explain the murmur a soft fibrinous vegetation is found on the surface of a valve. In some cases they, on the contrary, become firmly fibroid or pseudo-cartilaginous in aspect, or undergo calcification or pseudo-ossification.

771. Concretions of all the varieties, producible before death, once fixed to the endocardial surface, generally remain permanently attached; but separation is possible either in mass, in moderate-sized fragments, or in minute particles. The consequences of such separation may prove, to the last degree, grave.

772. *Causes.*—Whatever be the ultimate form assumed by a cardiac concretion, it originates simply in coagulation of the blood. In some cases the process is sudden and effected on a large scale—in others slow and of very limited extent.

The conditions promoting the necessary coagulation are referable to: (*a*) obstructed movement of the blood through the heart; (*b*) anomalous composition of the blood; (*c*) endocarditis.

(*a*.) Obstruction of the intracardiac current may result from all varieties of valvular impediment; from general dilatation or local pouching and aneurism; from mere roughness of surface, caused by fissuring or otherwise; and from pressure, especially on the right side of the heart, by mediastinal tumors or sacculations of the aorta. Although from the feebleness of contractile power of a fattily metamorphosed heart, stagnation, with sequential clotting of the blood, might be expected to be of frequent occurrence, it actually proves, so far as I have seen, to be rare. In exhausting diseases of various kinds, provided the fibrin be not seriously reduced in plastic quality, coagulation may take place towards the close of life.

(*b*.) Among the anomalous conditions of the blood favoring the clotting-process, foremost stands the hyperinosis of sthenic inflammations and certain specific affections. In rheumatic fever, croup, and especially in pneumonia (as originally insisted on by M. Bouillaud) the chances of serious obstruction in the right heart through the formation of non-soluble coagula add materially to the other sources of danger in those affections. Separation of the fibrin is also favored within the heart in certain forms of poisoning of the blood, as in common pyohæmia, or that sequential to glanders. In anæmia, just as spontaneous coagulation occurs in the veins of the lower extremities and in the sinuses of the dura mater, so is it occasionally, though rarely, effected within the heart.

(*c*.) However observers may vary in their estimates of the frequency with which noteworthy clotting occurs in acute endocarditis, the mechanism of its occurrence is plain enough. Roughness of surface, produced by deposition of lymph, and occasionally by minute fissuring; the hyperinotic crasis of the blood; the poisoned condition of that fluid arising from the inflammation-products, which are thrown into the general current from the endocardial surface; and the obstruction of circulation from valvular difficulty all contribute their share in effecting the result.

773. Coagula produce certain effects when fixed to the surface; they produce others through their separation in totality or in fragments therefrom.

1. Their effects, essentially obstructive when fixed, vary with their size, their precise situation, especially in respect of the orifices, and their rapidity or slowness of formation. Nothing can be more singular than the degree to which large masses may be tolerated within the heart, provided their growth has been a work of time. The case of huge encephaloid tumor of the right auricle, referred to in a former place [684], where no single cardiac symptom had ever occurred, until, from altered relationship of the mass to the orifices of the auricle, fatal obstruction was instantaneously produced, sufficiently illustrates the statement.

The singular manner in which a state of orifice, permitting reflux, may be converted by coagulum deposit into one of pure obstruction, is elsewhere proved [733].

If a coagulum be, as it were, semi-fixed *in situ* by a long pedicle, it may float in and out of an orifice, in such manner as now to obstruct the onward current, now to cause reflux [696, *a*].

2. If a coagulum of some size be separated from its attachments, constant risk of its becoming impacted in an orifice, so as to cause sudden and fatal obstruction, ensues. The fact has been observed at the mitral orifice.[1] Such detachment may take place while the coagulum is yet moderately soft, or after it has undergone more or less complete calcification.

More frequent is the detachment of minute fragments of soft clot or firm vegetation, which, borne into the blood-current, either pulmonary or aortic, eventually reach a spot, beyond which, from the narrowness of the vessel, their further progress becomes an impossibility. Under these circumstances one of two things happens; either, after having impeded the local circulation for a time, the minute coagulum breaks up, dissolves, passes on and leaves the interior of the vessel free; or the little fragment remains fixed as a permanent plug, exciting inflammation, coagulation, or hemorrhage, according to the special textural conditions of the spot.

774. *Physical signs.*—The physical signs of abrupt and extensive coagulation within the heart are on the whole sufficiently characteristic. Sudden irregularity in the impulse, affecting both force

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[1] Van der Byl, Lancet, January, 1837.

and rhythm; extension of percussion-dulness, mainly to the right of the sternum; obscurity of the normal sounds, especially of the first, or marked inequality in their strength with successive beats of the heart; impairment or increased intensity of a murmur or murmurs previously existing; or the production of a new murmur or murmurs, either obstructive, or reflux, or both. This variety in the conditions of murmur is easily explained by the various relationships which the coagula may assume in regard of the orifices, the valves, and the tendinous cords. The most common murmur is that indicative of obstruction in the infundibulum of the right ventricle and pulmonary orifice, systolic at and below the base and extending upwards to the left of the sternum. Greatly less frequent is the parallel murmur indicative of obstruction in the left ventricle.

775. *Symptoms.*—The symptoms of clot-formation, when occurring in its gravest form, in connection with endocarditis, have already been detailed [514].

In less urgent cases, both as regards the actual amount of coagulation and the condition of the heart prior to its occurrence, the symptoms are much less grave. An increase in the amount of pre-existing chest-symptoms, dyspnœa, dusky lividity of surface, distension of the external jugular veins, and increased frequency with variously perverted rhythm of the pulse. Spongy tumidness of the base of the neck, such as that very habitually significant of pressure on the large intrathoracic veins by tumor or aneurism, is sometimes observed; or actual œdema may take place in that situation—or, on the contrary, be first seen about the feet. Whatever amount of general distress may have pre-existed undergoes more or less marked increase.

Continual nausea and vomiting have occasionally been observed for several days before death, in persons whose hearts contained coagula evidently formed during life: they have been looked on as symptoms of coagulation. But the mode of connection of the two things is not clear; concretions may certainly form without disturbing the stomach, while unquestionably prolonged nausea will promote intracardiac stagnation and clotting of the blood.

776. Cessation of life, in cases clearly fatal through the influence of rapid and extensive clotting within the heart, generally follows very shortly on the process. I have known death occur in sixteen hours after the physical signs first distinctly indicated new intracardiac obstruction, proved *post mortem* to have depended on coagula. Life is rarely prolonged beyond the third day. But we must remember that the *onus* of destroying life is in these cases commonly shared by some grave pre-existing disease, endocarditis, pneumonia, &c. Where, on the other hand, the process is slow and on a limited scale, life may be prolonged considerably—nay, if the formation, even in certain situations of most perilous influence, be very slow, existence may not be demonstrably shortened by the occurrence.

777. *Diagnosis.*—The local signs and general symptoms of nervous shock to the heart, the local signs and systemic effects of intracardiac obstruction, *plus* the existence of the blood-states already enumerated [772], make up the groundwork of diagnosis in cases of extensive and serious coagulation.

It is almost unnecessary to say that in the characters of the murmur or murmurs, arising out of the obstruction of coagula, there is nothing specially distinctive. The circumstances, under which the murmur occurs, furnish the main guide to its mechanism. Its systolic rhythm and transmission, either in the line of the pulmonary artery or of the aorta, are obviously valueless as positive evidence.

Coagulation on the left, much less frequent than on the right side, differs symptomatically from the latter in its tendency rather to congest the pulmonary than the systemic capillaries. The difference is precisely the transcript of what is observed severally in cases of chronic mitral and tricuspid impediment.

In cases of acute endocarditis, I know of no means by which coagulation can with surety be distinguished from rupture of a valve or ulcerative destruction or rupture of a tendinous cord. The effects of cardiac shock are the same in all three cases; so are the physical signs identical; indeed this must be so, as clotting to greater or less amount will of necessity occur around the injured spot of the valvular apparatus.

778. *Prognosis.*—The prognosis of coagulation within the heart is, as far as existing knowledge guides us, grave in the extreme. In truth, it has not occurred to me to witness more than two or three cases where the diagnosis appeared to be solidly established, and where disappearance of the symptoms and physical signs, with present recovery of the patient, took place. And even here, as, I am thoroughly sensible, the diagnosis could not be esteemed wholly unopen to objection, my evidence of recovery is not as satisfactory as might be desired.

779. *Treatment.*—The measures to be adopted in cases of endocarditic concretions have already been described [519]. And these measures may be had recourse to in all acute inflammatory diseases, where coagulation occurs, especially on the right side of the heart. Where the left cavities are the seat of stasis, I should expect more advantage from free dry cupping of the chest generally, than from any other local agency. In chronic diseases with languid circulation, the risk of coagulation should be averted as far as possible by abstaining, at least in any quantity, from the use of nauseant medicines and digitalis, the obvious effect of which must be to slacken the circulation. Bloodletting should also be refrained from, unless under some urgent necessity. The influence of diluents, recommended for the purpose of rendering the blood less coagulable, can scarcely be trusted to—and by producing a watery plethora they may do more harm than good. Small quantities of

bicarbonate of potass, taken two or three times a day, seem to me the most promising prophylactic.

But the crasis of the blood must of course always be taken into consideration; the iron and fibrinous diet essential in spanæmia would be utterly out of place where a disposition to hyperinosis could be traced.

§ XIII.—MALFORMATIONS OF THE HEART.

780. There are a certain number of malformations of the heart and its appendages which, interesting enough to those who make special pursuits of morbid anatomy and teratology, possess little or no importance in a clinical point of view. We shall scarcely delay with the description of these.

I.—MALFORMATIONS UNATTENDED WITH SYMPTOMS.

781. Deficiency of the pericardium, the heart lying mainly in the left thorax, and at somewhat a lower level than natural, has several times been recorded.[1] In no one instance of which I have read the narrative, from the case of Baillie downwards, were any symptoms noticed. In some of the subjects a sort of rudimentary serous sac could be traced.

But though symptoms may be wanting, it seems probable enough the physical examination of the heart's motion might disclose something abnormal. There is no present attainable evidence on the matter.

782. Excessive congenital smallness of the heart would be attended with physical signs undistinguishable from those of acquired concentric atrophy [580]. The state is certainly unproductive of local annoyances of any kind; and though it has been seen in connection with epilepsy, the relationship of cause and effect between the two things may reasonably be doubted.[2]

783. Perversions of shape, at least anatomically curious, have occasionally been noticed. Thus the apex has been found bifid.[3]

784. The valves are subject to three main forms of congenital imperfection. Deficiency of number, excess of number; malposition, with deficiency of structure, the tongues of the affected valve, largely perforated, lying across the blood-current.

There cannot be a doubt, certain of the conditions falling under these heads must be productive of abnormal physical signs, though, what these may precisely be, experience has not yet taught us. Of subjective effects, if such occur, nothing is yet known.

Were physical signs to occur in connection with the malformed semilunar or auriculo-ventricular valves, they must of necessity be significant either of obstruction, of reflux, or of both; nor can I imagine the possibility of distinguishing such mechanism from that of the murmurs of acquired disease. Yet there are contingencies under which such power of distinction would be practically useful; as, for instance, in individuals laboring under certain diathetic affections (rheumatism, Bright's disease, &c.), prone to engender endocarditis—and who are found to present a morbid sound bearing all the attributes of a murmur caused by inflammatory changes, but nevertheless, it might be, really resulting from congenital malformation.

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[1] Univ. Col. Museum, Carswell's Drawings, A., No. 446—here the fibrous layer only wanting.

[2] Nasseau, Arch. Gén. de Médecine, t. viii. p. 603.

[3] Parise, Bulletin de la Soc. Anat., 1837.

Dr. Peacock has shown that many of these valvular malformations lay the foundations of disease in after life.[1]

II.—MALFORMATIONS ATTENDED WITH SYMPTOMS.

CYANOSIS.

785. There are on the other hand a considerable number of malformations of the heart which, generally speaking, not only prove more or less incompatible with prolonged extra-uterine life, but also, while that life continues, produce more or less grave symptoms. Among these symptoms, though it yields to many others in real importance, a certain bluish or livid discoloration of the skin has always attracted most attention—to such a degree, that this symptom, with its functional accompaniments, has been by the nosologists elevated to the rank of a specific disease, under the titles of cyanosis, or morbus cæruleus. And however indefensible scientifically this mode of treating the subject may be, it carries with it so many practical advantages that we will not depart from it here. Let us first, then, review the group of clinical conditions characterizing cyanosis.

786. *Symptoms.*—The discoloration of the tegumentary membranes, especially the skin, may be referred to two main types—blue, and pale claret. As will hereafter appear, this distinction is probably not unimportant. The blue color ranges between various shades of light or dark leaden, purplish, or almost black; the claret tint may be very light, or of the deepest shade that is ever seen in *post-mortem* vibices, or even in rubeola nigra.

The discoloration may pervade the whole surface, but attains its maximum at the internal canthi, lips, nose, ears, below the eyes, about the genital organs, and at the tips of the fingers and toes. Changeable in intensity from time to time, the tint greatly deepens from palpitation, effort, fits of ill temper, dyspnœa, and, in a word, from all conditions adding any functional obstacle to the statical difficulty of the circulation already existing. So long as respiration remains easy, it is undeniable that in some instances the tegumentary color scarcely deviates from the natural hue. Generally speaking, the color, of whichever type it be, lightens after death.

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[1] Malformations of the Heart, p. 95.

The ends of the fingers, if life continue for any length of time, grow more or less bulbous; in adults this peculiarity of form is sometimes singularly well-marked;[1] at the same time the nails grow incurvated; and the patients are said to be particularly subject to whitlow, and to sluggish cutaneous sores about the mucous orifices.

The temperature of the surface and mucous canals, at their outlets, is low; the patients, constantly chilly and sensitive to the least fall in the thermometer, will bear a large fire in the warmest weather this climate affords. Œdema of the feet sometimes exists. The frame is weak, the muscular and adipose systems ill-nourished, sometimes peculiarly wasted. Nevertheless, even where cyanæmia has reached the extremest limit, where the whole system derives its sustenance from dark blood, the viscera may be of full size, and in point of color present no serious difference from healthy organs. At least such I found to be the fact in a case where the aorta rose from the right, and the pulmonary artery from the left, ventricle, while the great veins held their normal relationship to the right and left auricles, the only medium of communication between the two distinct circulations, capable of conveying florid blood to the systemic capillaries, being the ductus arteriosus. And in this case, as the blood in the portion of the aorta lying between the opening of the ductus arteriosus and the heart was wholly venous, and as the coronary arteries rose from the aorta in the usual position close to the sigmoid valves, it follows the nutrition of the heart, which was hypertrophous, must have been wholly effected by non-oxygenated blood.[2] Possibly the slight degree to which the color of the viscera differed from that of health may have depended upon their tint, as well as that of the tegumentary membranes, having lightened after death. M. Louis states that the color of inflamed organs is the same as in persons with naturally colored blood.[3]

The digestive faculty is habitually feeble; and in a certain number of cases more or less persistent diarrhœa has contributed to enfeeble the system or even destroy life.

Though rarely the subjects of serious habitual dyspnœa, cyanotic persons, with the rarest exceptions, suffer from paroxysmal difficulty of breathing—often the result of disturbed circulation, caused by fits of petulance and passion, to which they prove, above the average, prone. Cough, either dry or attended with watery expectoration, probably through œdema of the lungs; frequent attacks of congestive bronchitis, added to, sometimes, the physical signs of emphysema, or of atelectasis, congenital or acquired, or of all three combined; constitute the sum of pulmonary symptoms and morbid states.

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[1] Occasionally pushed to greater extremes than I have ever seen in cases of empyema—*e. g.*, Univ. Coll. Museum, Drawings, A., No. 711.

[2] Medico-Chirurgical Transactions, vol. xxv. Nasse affirms the spleen, thyroid gland, and supra-renal capsules are particularly small; but the contrary has been noted.

[3] Communication des Cavités Droites et Gauches, p. 340.

In many cyanotic cases the heart is habitually quiet enough; but paroxysmal disturbance of its action is common to them all. Palpitation active, tumultuous, with strong diffused impulse, a pulse ranging from 120 to 160, irregular in force and rhythm, and sometimes almost imperceptible, commonly coexists with the dyspnœal paroxysms above referred to; syncopal tendency and actual syncope, though rare, do sometimes actually occur; the eyes are prominent, the expression wild, and the arms tossed violently about. The duration of these fits ranges from a few minutes to some hours; and they may be of frequent, daily, nay, almost hourly, occurrence, or reappear at rather rare intervals. There is no particular pulsation observable in even the most deeply-colored parts, though the capillary vessels are sometimes distinctly enlarged.

Intellectual activity and aptitude for acquiring knowledge rank low in cyanotic children: deficiency of this kind has been almost invariably observed when the child has survived sufficiently long. The congestion of the brain and cord, arising in the quasi asphyxial state of the fits of dyspnœa, often issues in convulsions, somnolence, and even temporary coma. Reflex phenomena, such as jerking action of the limbs and grinding of the teeth, are frequent during sleep.

Cyanotic children usually exhibit a sad, suffering expression of countenance; decrepit, and prematurely old-looking, they care little for the ordinary amusements of their age, dreading the occurrence of a paroxysm through the slightest exertion or exposure to chill.

787. *Physical signs.*—The conditions in a case of cyanosis which may conceivably give rise to morbid physical signs, are the cyanæmia itself, the malformation, and the disturbed dynamism of the heart generated by both.

Now, does cyanæmia induce murmur? Is it possible that the constitution of dark blood is *per se* a sufficing cause of soniferous movement, and that herein lies the hitherto ignored, or at least one of the ignored, causes of "venous murmur" [293]? The answer must be in the negative; for in the most intense form of cyanosis, where the whole system is fed with very little else than black blood, as in cases of the arterial transposition above referred to, there need not be, and is not, of necessity, any murmur at all audible in the cardiac region.

Next, as concerns the malformation, the physical signs indicated must, of course, vary with its precise nature; and it is remarkable enough that, even in the gravest varieties (take, again, as an instance, the arterial transposition), the structural elements of murmur may be wholly wanting.

Thirdly, few malformations, even aided by the morbid crasis of the circulating blood, maintain persistently a state of dynamic disturbance of the heart. Increased impulse of disturbed rhythm is essentially a paroxysmal occurrence.

It seems advisable to warn the student, farther, that none of the malformations producing cyanosis, of themselves, alter the areas of deep or superficial cardiac percussion-dulness. If those areas be increased, sequential hypertrophy or dilatation, or both, have been at work.

In the most common variety of malformation connected with cyanosis, namely, an open state of the foramen ovale and constriction of the orifice of the pulmonary artery, with sequential hypertrophy, simple or eccentric, of the right ventricle, systolic basic thrill, doubtless in the pulmonary artery, may be felt; the systolic murmur of pulmonary constriction may be traced up to, about, and beyond the second left cartilage; and the signs of an hypertrophous state of the right ventricle will be coincidently found. I do not know that we are in a position to assert positively, that patency of the foramen ovale may ever, of itself alone, cause murmur; for in all records I have met of the coexistence of the two things, either constriction of the pulmonary orifice is actually stated to have existed, or it may have existed—all mention of the vessel being omitted by the narrator. On the other hand, no satisfactory evidence exists that patency of the foramen proves invariably, and under all circumstances, incapable of inducing murmur. It is, of course, certain the foramen may be even large enough to admit the point of the little finger, without a particle of murmur occurring;[1] but, *per contra*, it is very well conceivable that the tension, or other conditions, of the edges of the opening might, in particular cases, lend themselves, with unusual ease, to sonorous vibrations. Strength of current, we know by experience, is not, under otherwise favoring circumstances, requisite for the production of murmur. And Dr. Hughes Bennett[2] suggests, and Dr. Markham actually maintains, he has heard a loud murmur thus generated. In a slightly cyanosed child, aged four years, a rough systolic murmur, therefore coincident with the diastole of the auricles, audible about the heart's base and the left infra-clavicular region, indistinct below the nipple, scarcely to be caught at the heart's apex, loudly defined at both sides of the upper half of the interscapular space, seemed inexplicable, *post mortem*, except as the result of struggle of the blood at an open foramen ovale. The communication from the right to the left auricle was sufficiently large to admit the point of the little finger; and there was no constriction of the pulmonary artery, nor any other condition of heart, conceivably explanatory of the murmur.[3] The precise time of an intra-auricular murmur may be made matter of dispute; but, admitting with Dr. T. Fox,[4] that the moment ought to have been pre-systolic, is it not very likely a murmur really of that rhythm might have been mistaken for one of systolic time? Dr. Fox suggests, by inference, that the murmur might have depended on coarctation of the arch of the aorta; but in time it ought then to have been post-systolic.

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[1] For example, my case, Med.-Chir. Trans., already referred to; patient ætatis 10 months.

[2] Clinical Medicine.

[3] Brit. Med. Journal, 1857.

[4] Med. Times and Gaz., Sept. 1859.

Cyanotic murmurs are sometimes exceedingly loud. I have known a murmur of this kind audible at a small distance from the surface.

783. *Period of Origin and Duration.*—Cyanosis is far from being always congenital. Of 101 cases of malformation, attended with well-defined symptoms, in 74 these were noticed at or very shortly after birth; in 15 they appeared before the end of the first year, and in the remaining 12 at various periods, from ætatis 1 to 14.[1] Stillé's return gives 40 congenital, 31 non-congenital, in a total of 71.[2] There cannot be the smallest doubt, however, that in many cases where the symptoms have first attracted attention at an advanced period of childhood, they had not then come into existence for the first time, but merely undergone striking increase.

It has been suggested, by different classes of theorists, that when cyanosis is non-congenital, and attracts notice for the first time in early childhood, enlargement of the heart gradually opens out the foramen ovale, and so induces the intermixture of venous with arterial blood; or that congenital constriction of the pulmonary artery increases. In the adult, the appearance of cyanosis has sometimes been apparently traced to a blow, a fall, or an effort; possibly some forcible separation of the edges of the foramen ovale had occurred: sometimes ulcerative openings in the auricular or ventricular septum have seemingly proved the immediate cause. Occasionally the external evidences of the malformation have been but slight, until the accidental occurrence either of endocarditis or of serious bronchial or pulmonary inflammation: the mechanism in these two cases is, of course, different.

Once established, cyanosis, as a rule, to which there can be but few exceptions, remains a permanent condition, though its exact amount may vary from time to time. Dr. T. Fox ably argues, on the evidence of the following case, that cyanosis may, under special circumstances, be temporary. A child became very darkly cyanosed on the eleventh day after birth—no other cyanotic symptom existed, and no abnormal sound was detected at the heart. In forty-eight hours the intensity of the discoloration had yielded; in a week no trace of it remained, its disappearance having been attended with the changing hues of ecchymotic absorption and slight jaundice. Dr. Fox tenders the following explanation: the ductus arteriosus and foramen had become on the eleventh day, that of the appearance of the discoloration, less and less patent—but probably advancing to complete close with less than usual rapidity. By the closure, first of the duct, then of the foramen, all the blood of the right side of the heart was compelled to travel through the pulmonary artery, which now presented some "obstruction," partly congenital, partly due to the lateness of the normal changes in the duct and foramen; after a little time the "obstruction" was overcome, and the cyanosis removed.[1] But if the postulated "obstruction" existed, why was there no murmur; why were there neither dyspnœa nor cardiac distress? Jaundice, vomiting, serious hæmaturia, and, above all, extravasation of blood into the skin, shown by the changing hues of the hæmatin, as described, are strange accompaniments of cyanosis. Altogether the case is singularly mysterious.

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[1] Peacock, loc. cit., p. 118.

[2] Amer. Journ. Med. Sci., N. S., vol. viii., 1844.

789. Laennec had a notion that cyanæmia in some measure antagonizes tubercle; Rokitansky, pushing the idea to extremes, has taught that cyanosis, like other states in which venosity of the blood predominates, "offers a complete protection" against tuberculization. Several cases are on record showing the fallacy of this doctrine absolutely stated;[2] what degree of antagonism, if any, really exists between the affections, has scarcely been submitted to statistical investigation. As far as such inquiry has gone, it tells wholly against the antagonistic theory; for Dr. Peacock finds evidence of active tuberculization in 16.07 per cent. of cases in which the patient, having malformed heart and cyanosis, survived the age of eight years.[3]

Cyanosis seems to have some share of influence in producing pericarditis: but this point also requires further examination.

Bizot has shown, in refutation of an old idea, that the foramen ovale is somewhat more frequently closed in phthisical, than non-phthisical persons.[4]

790. *Manner of Death.*—Death is usually of slow asthenic type, through imperfect hæmatosis—facilitated in many instances by pulmonary atelectasis, congenital or acquired, and emphysema. Or cerebro-spinal phenomena of congestive character may abruptly destroy life; serous effusions and visceral hemorrhages also hasten the fatal issue occasionally. These are almost essential effects of the disturbed condition of the circulation; but accidentally death may occur from almost all varieties of acute or chronic disease, in patients who survive the period of early childhood. In some instances the manner of death has not only distinctly been phthisical, but of ordinary phthisical type.

791. *Anatomy and Mechanism.*—The symptomatic state just described is known by experience to be connected with various malformations of the heart and perverted modes of origin of its great vessels. The general tendency of the more common of these malformations is to alter the relationships naturally subsisting

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[1] Med. Times and Gaz., August, 1859.

[2] *e. g.* Louis, loc. cit., p. 313; and Fearnside, Association Journal, March, 1854. Dr. Chevers has collected thirteen similar cases (Pulmonary Artery, p. 136).

[3] Loc. cit., p. 137.

[4] Mém. de la Soc. Méd. d'Observation, t. i. p. 360.

between the two sides of the heart and the two kinds of blood—dark and florid. But some of the number act in other ways, and the entire series may be referred, as species, to three classes, as follows:—

A. *Conditions permitting direct communication of the arterial and venous circulations.* (*a.*) *In the heart.*—Open foramen ovale; congenital deficiency of part of the ventricular septum; acquired perforation, of ulcerative or other character, throwing the auricles, or the ventricles, or all the four cavities into, practically speaking, a single cavity; heart formed of one auricle and one ventricle, the latter giving off one artery, which divides into a pulmonary artery and aorta, &c. (*b.*) *In the great vessels.*—Freely pervious ductus arteriosus; aorta rising from both ventricles, or from the right ventricle, or from a trunk common to itself and the pulmonary artery, &c.

B. *Conditions causing distribution of black blood almost solely to the systemic capillaries, and of red blood to the pulmonary capillaries, without, practically speaking, any intermixture of the two kinds of blood.* Here appear cases where the aorta rises from the right, and the pulmonary artery from the left, ventricle—the venæ cavæ, as in the natural state, communicating with the right, and the pulmonary veins with the left, auricle;—where, consequently, there are two distinct circulations, communicating alone by the foramen ovale and ductus arteriosus, if (as is the rule) these remain open.

C. *Conditions obstructing the entry of blood into the lungs or intensely congesting them, so as to prevent oxygenation.* (*a.*) *In the heart:*—Excessive smallness of the right ventricle; extreme narrowness of the tricuspid orifice: these states disturb the process of distribution of blood *to* the lungs. Great contraction of the cavity of the left ventricle, great coarctation of the mitral orifice; these conditions prevent the return of blood *from* the lungs. (*b.*) *In the great vessels:* Partial or complete obstruction of the orifice of the pulmonary artery.

Two different theories have commonly been urged in explanation of cyanosis as a dependence on these abnormal conditions:—(1.) The venous and arterial intermixture theory; (2.) The systemic venous stasis theory.

(1.) The open condition of the foramen ovale lending itself to intermixture of the two currents, and, in consequence of frequently coexisting hypertrophy of the right auricle, facilitating flow from the right to the left side, rather than in the converse direction, seemed to afford a ready explanation of the dark tint. This theory of intermixture was long received without question; but against its exclusive adoption the following facts depose emphatically: 1. Free communication may exist between the blood-currents, as far as the existence of a widely gaping foramen proves this, without cyanosis. 2. Cyanosis has been wanting where there was but one ventricle, and where the aorta rose wholly from the pulmonary artery. 3. When cyanosis coexists with the anatomical conditions of intermixture, no direct ratio holds between the amount of discoloration and the freedom of communication. In a case noted by M. Louis, where the foramen only equalled a lentil in size, and the orifices and cavities of the heart proved free, more discoloration had existed than in another where the opening was much larger, and the pulmonary orifice contracted.[1] 4. Ribes has related a case where the aorta rose from the right ventricle, and yet cyanotic symptoms did not appear until the age of three years. 5. Fouquier observes that the fœtal skin, though always circulating black blood, is not cyanosed.[2] 6. In the natural state the color of the skin comes of the different colored bloods moving in capillaries and venous radicles lying in close juxtaposition; the effect must be the same as if different bloods were mingled in the same vessels: therefore, as the former condition does not cause cyanosis, there is no reason the latter should do so.

(2.) Morgagni started the opinion that cyanosis is really to be ascribed to systemic stasis in the venous radicles, arising from obstruction at the pulmonary orifice. M. Louis supports this view. He observes that the blueness of the fore-arm, when ligatured for venesection, does not come of want of arterial blood, for this continues to reach the textures, but from engorgement of their venous system. Stillé, arguing in the same direction, notes more or less grave obstruction of the pulmonary orifice in fifty-three of sixty-two cases. Abnormal septum in the right ventricle, exercising a like obstructive influence, is attended with cyanosis.

But this explanation, too, proves inadequate. For:—1. No direct ratio holds between the amount of cutaneous discoloration and the amount of narrowing of the pulmonary orifice. M. Louis has himself incidentally conceded this, as we have just seen. 2. This holds true, whether the pulmonary obstruction be acquired or congenital. Nay, more, a case observed by Dr. H. Roe shows there may be no cyanosis at all, from even great congenital contraction of the vessel. 3. How is this doctrine reconcileable with the fact, that the most intense venous obstruction, intra-cardiac or extra-cardiac, but within the thorax, may occur without inducing true cyanotic discoloration? In the course of my experience I remember to have seen but one case of thoracic disease—the gravest example of emphysematous atrophy of the lung I ever observed—in which the tint of skin was fairly assimilable to that of really deep cyanosis.[2]

It stands, then, an unassailable truth, that the foramen ovale may be widely patent without the occurrence of cyanosis; so, too, the fact is equally well established, that the pulmonary orifice may be congenitally constricted to a high degree, and the skin be of natural color. But a satisfactory *tertium quid* may, I think, be moulded out of the two theories. Grant that perforated septum and constricted pulmonary orifice coexist, and the occurrence of cyanosis becomes a certainty; on the one hand, the labor required at the right side of the heart to overcome the obstacle at the pulmonary orifice, forces of necessity venous blood through the foramen;[1] while, at the same time, the systemic venous stasis, resulting from the pulmonary obstruction, contributes to darken the tint. Probably, too, that tint will prove of the blue or of the claret variety, according as intermixture or stasis severally predominates.

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[1] Communication des Cavités, &c., p. 344.

[2] *Vide* Appendix.

The effective power of intermixture, provided the venous quota be sufficiently large, is clearly proved by cases of transposition of the great vessels, such as that already more than once referred to. In this case the surface was permanently leaden-blue in tint, the only arterial blood reaching the systemic capillaries being that carried through the ductus arteriosus. No mechanical obstruction existed in the heart; but the hue grew notably darker under all dynamic influences, impeding the circulation through the lungs. In this instance, too, a slightly corrective condition of the intense cyanæmia arose out of the relative calibre of the great vessels at their origin, as explained in the history of the case (*loc. cit.*, p. 12); and it appears to me exceedingly probable, analogous arrangements (as of the bronchial arteries, for instance), indirectly promoting arterialization, may exist in many cases without being detected.

792. *Causes.*—Defective development of the heart is considerably more common in males than females: according to a calculation by Dr. Peacock, in the ratio of 57.2 to 42.8. The explanation remains to be found. Malformation of the kind is sometimes hereditary. All physical defects in her offspring are invariably referred by the mother to influences, mechanical or emotional, occurring to herself during pregnancy; but there is no evidence approximating even to proof, that congenital vices in the heart are thus produced.

To fœtal endocarditis may commonly be traced obstruction at the pulmonary orifice; certain valvular affections are similarly engendered during intra-uterine life. For the history of errors of development I must refer to works on Teratology.

793. *Diagnosis.*—(*a.*) In the new-born infant the only affection with which it appears possible to confound cyanosis, is apoplexia neonatorum (intense congestion with actual extravasation of blood into the membranes of the brain and spinal cord), clinically characterized by great lividity of face, swollen scalp, feeble action of the heart, slow, irregular respiration, clenching of the hands, convulsive actions, torpor, chilliness—conditions lapsing into fatal asphyxia,

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[1] If the pressure of the current on either side of the opening be equal, there is no reason why each current should not pass on without commingling at all, or more than very slightly, with the other. Such nicely-balanced pressure doubtless exists in those numerous cases of open foramen, unattended with cyanosis, where there is no constriction of the pulmonary orifice, and no dilated hypertrophy of the right cavities. It is almost self-evident that the admixture must be very free to modify perceptibly the color of the blood.

unless treatment, especially bleeding from the umbilical cord, prove successful. But the tint of skin in cyanosis is different, bluish, not livid; the scalp is not swollen, nor is there general tumidness of the upper part of the body; the action of the heart is rather in excess than deficient in strength; and the respiration is not labored, irregular, and slow.

(*b.*) In the young adult the distinction is more difficult. It may well happen that a slight amount of malformation, insufficient in itself to produce cyanosis, may have existed at birth, and eventually proved the cause of blue discolorations, either through its own increase or through the contingent aid of some acquired obstructive disease of the organs of circulation or respiration. How is cyanosis of this mechanism to be distinguished from discoloration wholly due to acquired disease? Marked blueness, rather than lividity of tint, will depose in favor of partially congenital origin; but a positive diagnosis will prove unattainable.

(*c.*) The specific vice of form, affecting the heart or vessels, can in some varieties be at least strongly surmised, if not actually announced with certainty. Thus, narrowing of the pulmonary orifice with a defective septum, may be diagnosed with much security, when a systolic basic thrill, with murmur, transmitted to the left and upwards, and scarcely audible posteriorly [214], coincides with well-defined cyanosis.[1] As life advances, the conditions of right-sided hypertrophy would further strengthen the diagnosis.

Again, if a child were deeply cyanosed at birth, and no murmur could be heard, we should be justified in strongly suspecting that the aorta and pulmonary artery were transposed; or that some other grave vice, throwing no physical obstacle in the way of their blood-currents, affected the great vessels at their origin.

794. *Duration of life and prognosis.*—The duration of life varies with the nature of the malformation: but the degree rivals in importance the form of the vice in the heart. And experience amply shows that the issue depends also on extrinsic conditions, independent of form and degree of malformation. Hence there is no security of prognosis. An infant with moderately constricted pulmonary artery may perish almost at birth; while, it is certain, life may be prolonged to the twelfth year, though the vessel be absolutely impervious. Again, recorded cases of transposed great vessels show the malformed child may struggle on to the near completion of the third year, or perish within the first few days of extra-uterine life.

Some practical guidance may be had from the degree of frequency of the suffocative paroxysms.

795. *Treatment.*—The treatment of a case of cyanosis resolves itself into the prevention, as far as possible, of paroxysms of dyspnœa and palpitation. Tranquillity of the circulation, by the avoidance of all emotional excitement, mental or bodily, and of all con-

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[1] See a valuable illustration by Dr. S. T. Speer, Med. Times and Gaz., Oct. 1855.

ditions likely to congest the lungs, the liver, and the abdominal organs, is to be aimed at; the temperature of the skin maintained by warm clothing, moderate exercise, and friction; and that of the body, generally, raised, if the stomach be not disordered thereby, by the free consumption of oil, fat, gum, and other aliments of respiration.

According to the testimony of Gintrac, alcoholic fluids produce drunkenness with unusual rapidity in, and are otherwise very injurious to, cyanotic persons: this is easily intelligible as a result of their imperfect respiration. The fact reminds one of the observing landlord, who noticed he could at will alcoholize his guests with small or large quantities of wine, according as he ventilated their dining-hall ill or well.

Where this is attainable, a warm dry climate should be resorted to.

§ XIV.—INJURIES AND WOUNDS OF THE HEART.

796. Foreign bodies, more particularly needles, sometimes enter the tissues of the heart, either directly through the chest-wall, or from the œsophagus.

797. In a case of the former kind the course of the needle was clearly marked out by the symptoms. It first entered the right mamma; a month later, while the patient was stooping to reach something from the floor, it penetrated more deeply, causing pleurisy; four months later she had pneumonia and bronchitis of the right lung; a month afterwards "spasms of the diaphragm," which were succeeded by obstinate vomiting and subsequently by pain about the heart and pericarditis. The needle was found after death, reaching from the right ventricle into the left.[1]

On the other hand it would appear a needle, at least five centimètres long, may be fixed in the ventricular septum, protrude into each ventricle, and be coated with fibrinous concretion at both ends without giving rise to any symptom. It is even affirmed no murmur could be heard, in the case specially referred to—a statement the more remarkable, as there was fibrinous exudation on the pericardial surface.[2]

So, too, balls have been known to lodge in the walls of the heart, and even protrude into the cavities, without inducing symptoms.

798. The subject of wounds of the heart is so commonly looked on as a part of surgical pathology, that a very few observations only will be ventured on here.

799. The symptoms vary with the character and direction of the wound, though not to so great an extent as might be anticipated: pallor, faintness, or complete syncope; uneasiness or actual pain about the site of the wound, increased by inspiration, and rather a dependence on the injury to the external soft parts than to the

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[1] Leaming, Br. & For. Med. Rev., July, 1845.

[2] Piorry, Union Méd., 1858.

heart's own structure [317]; external hemorrhage, very slight—or more serious, if an intercostal artery be wounded, or if a large external opening coincide with effusion of blood into the pericardium, effected prior to clotting in the actual wound of the heart itself; general anxiety, clammy perspiration, and other symptoms of collapse. The collapse is probably due in part to nervous shock, in part to actual loss of blood. The pulse may remain regular, though notably more frequent than natural; a normal pulse-respiration ratio of 4 : 1 may be maintained.[1]

If effusion of blood occur into the pericardial sac to any extent, its presence will be revealed by the sudden supervention of the physical signs of fluid accumulation, especially of the peculiar triangular-shaped percussion-dulness already fully described [107]. Friction-murmur will be inaudible, though a peculiar rumbling noise may be detected.[1]

But though the diagnosis may in the majority of instances be established on the evidence now briefly reviewed, taken in conjunction with the character of the external wound, the depth to which the cutting instrument may have penetrated, &c., still occasionally the utmost difficulty may be felt in determining whether the heart's fibre have really suffered or not. On the one hand cases are recorded where many of the symptoms indicating actual wound of the organ existed, yet it had wholly escaped; and on the other hand, as a single example of the negation of direct evidence, a case recorded by Dupuytren may be referred to. In this instance a man survived the infliction of several penetrating wounds of the right ventricle for upwards of three weeks, the pericardial sac meanwhile containing a large quantity of blood; and yet neither general collapse, nor any single local symptom, pointing to injury of the heart, occurred. But significant physical signs must, it would seem, have existed under the circumstances.

800. In the treatment the careful avoidance of too free stimulation on the one hand, or too lavish use of antiphlogistic measures on the other, seems to be the chief matter for consideration. On the whole less danger is to be apprehended from primary collapse, than from consecutive fever and some forms of cardiac inflammation.[2] Rest is all-important, and should be constantly maintained for days and weeks after all apparent local effects have passed away. From inattention to this rule, sudden fatal syncope has been known to occur upwards of three weeks after the accident.

Cardiac inflammation must be met by the cautious use of antiphlogistic remedies. Mercurials are recommended.

If extravasated blood stagnate, especially with added serous effusion, within the pericardium, paracentesis seems a reasonable measure; the results of the operation in so-called scorbutic pericarditis, indeed, justify a hopeful view of its probable success [548].

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[1] Boswell Reid. Lancet, Dec. 1855.

[2] Purple, New York Med. Jour., May, 1855, p. 420.

DISEASES OF THE GREAT VESSELS.

SECTION I.—AORTA.

§ I.—SIMPLE PULSATION.

801. AORTIC pulsation, or abdominal or epigastric pulsation or palpitation, as it has been variously called, is a peculiar functional affection of the aorta, essentially constituted by more or less throbbing action of the vessel. Although very positively observable in the thoracic aorta, it is best known in the abdominal division of the vessel.

802. *Symptoms.*—Pulsation at the epigastrium, more or less constant, but aggravated by various influences, such as brisk movement, nervous excitement, irritation of the bowels, or constipation and dyspeptic disturbance, is, as its title indicates, the prominent feature of the complaint. In rare instances, complete intermission of the throbbing action may occur from time to time—a fact important in respect of diagnosis. When strongest, the pulsatile movement is attended with a feeling of sickness or faintness, or pseudo-globus; there is no actual pain experienced, except the adjacent parts be accidentally extra-sensitive. The epigastrium, however, always bears pressure worse than in healthy people, and may be extremely tender.

The constitutional effects of aortic pulsation are sometimes sufficiently serious; it increases already existing nervousness, excites apprehension, or obstinate conviction, on the part of the patient, of deep-seated organic disease; deprives him sometimes of the power of taking exercise, and interferes with digestion, whence arise loss of appetite and emaciation: and some patients, constantly dwelling on the symptom, magnifying its importance, and, fancying that bystanders must notice it as well as themselves, acquire an utter distaste for all society.

803. *Physical signs.*—In well-marked cases, pulsation may be easily seen at the epigastrium, especially in thin people; rarely at the umbilicus. The hand, laid on the surface in the course of the vessel, receives a forcible forward impulse, slapping rather than heaving, jerking, quick, abrupt, without distinctly expansile character, but bounding and free—varying in degree from an action so trifling as to be scarcely perceptible, to one sufficiently powerful to shake the bed. As a rule, there is no lateral expansion to be

felt; but, if hardened tissue lie on the confines of the vessel, such expansion may at the least be closely simulated. Unless the blood be anæmic, no thrill is to be caught. The vessel, if the patient be very thin, may be reached on either side by the fingers, and slightly moved laterally. The transverse limits of the vessel under percussion are natural; but it must be confessed this is a fact very difficult of establishment: the abdominal wall should first be steadily depressed for a minute or so, all gas and fecal matters, as far as possible, pushed sidewards, and then the vessel carefully percussed. Aortic pulsation may exist in the highest degree without murmur—a single systolic[1] impulsive sound being alone audible; or a systolic and a diastolic sound may be heard, the latter the fainter greatly of the two. Or, what is more common, a single systolic blowing murmur, prolonged slightly, rough and sharp, whiffing or whipping in quality, is heard—the more marked, the greater the pressure exercised on the front of the vessel. Such murmur may, however, in rare instances, be heard in the back. Until lately, I supposed that the discovery either of double or of diastolic murmur, in the course of the aorta, positively indicated disease in its coats; but I now know, by a case observed with great interest during life, that there may be intense aortic pulsation, with diastolic murmur, though the vessel is perfectly free from disease, and in calibre below the average. In the instance referred to, however, the pancreas was found, after death, enlarged and hardened, and had pressed somewhat on the vessel in the site of the murmur.[2]

804. It has been argued by some persons, on the ground of the limitation of the abnormal pulsation to certain spots of the vessel, that local organic change must exist. As to positive evidence, there is but little collected. In some of the few *post-mortem* accounts recorded, affirmation is absolutely made that the vessel was in all respects sound; in others, that its walls were flaccid (but as cause or effect of the pulsation?); in yet others, thin. I know from observation it may be texturally sound—and even one negative case is invaluable in a question of the kind.[3] Pressure from adjacent parts, if sufficient to reduce the calibre of the vessel, seems very distinctly to promote its undue action. I have known the pulsation change in precise site from day to day almost; and in rhythm the local throb differs distinctly sometimes from that of the heart and of the general tract of the vessel itself. All this points to purely dynamic essence; and the affection seems to consist of an excited state of local motor-innervation, commonly of reflex mechanism. It would be unjustifiable to deny the probability that a morbid state of the vascular coats may occasionally act as the first link in

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[1] In applying the terms systolic and diastolic to mark the rhythm of sounds and murmurs in the arteries, I intend to signify synchronism with the systole and diastole of *the heart*, unless the contrary be stated expressly.

[2] Gosling, U. C. H., Females, vol. v. p. 130.

[3] Gosling, U. C. H., *loc. cit.*

the chain of that reflex mechanism; I have seen well-marked atheroma in a case of the species.[1] But, on the other hand, there is no necessary connection between such local arterial disease and morbid pulsation; every day's experience exhibits grave calcification of the abdominal aorta, in persons who have been perfectly free from the complaint.

In some instances the entire of the abdominal tract, the iliac and femoral arteries are involved.[2]

805. The predisposing and exciting causes of aortic palpitation are sufficiently various. While it is not so common in ordinary hysteria and spinal irritation as might be expected, it accompanies many utero-ovarian diseases, with pelvic and abdominal neuralgiæ; follows anæmia of all modes of origin; occasionally attends plethora, sthenic and asthenic; and is induced in some susceptible frames, by green tea, strong coffee, tobacco, and similar agents. Various disordered states of the chylopoietic viscera produce it—simple dyspepsia, flatulence, and hepatic disturbances among the number; but special proneness to the complaint probably exists, where causes so slight suffice for its generation. Acute inflammation of the stomach, bowels, and peritoneum, as insisted on by Dr. Stokes, and chronic gastritis, as chiefly illustrated by Dr. Faussett, sometimes produce very marked sympathetic action of the abdominal aorta. Pressure of all kinds directly on the vessel takes an important place among its causes; I have known an accumulation of feces in the transverse colon induce it in an aggravated form.

806. Aortic palpitation, once fairly developed, is with difficulty got rid of. Baillie was probably right in the opinion that, though it might vary in amount, it seldom disappeared altogether. The chances of complete cure are greatest, where anæmia lies at the root of the evil; but even here there is no certainty of such result.[3]

807. *Diagnosis.*—A very little care will distinguish pulsation of the aorta in the epigastrium from the epigastric pulsation of a displaced heart, or an enlarged right ventricle: it is unnecessary to dwell on their distinctive marks.

The real difficulty consists in ascertaining with positiveness whether a given condition of abdominal pulsation be purely functional or dependent on a dilated state of the vessel. Now, first, if genuine sacculated aneurism exist, the pulsation is expansile, heavy, powerful, slow, and appears kept back, as it were, by some restraint behind it, instead of bounding freely forwards; thrill is occasionally to be felt, and a tumor, with dulness under percussion nearly commensurate with its size, is discovered. A harsh, grating, hollow murmur, systolic, of the characters elsewhere described, audible in front, may also sometimes be detected along the spine posteriorly. All this is accompanied with pain more or less wearing or agonizing; and the determination of the true nature of the case proves, as a rule, sufficiently easy. But, secondly, to distinguish between incipient fusiform dilatation or sacculation on the one hand, and dynamic pulsation on the other is, I believe, often matter of impossibility. A man aged about thirty was sent to University College Hospital by Dr. Siordet, for an opinion on the nature of the epigastric pulsation under which he suffered. So nearly balanced was the evidence, that I did not venture to pronounce an opinion in one direction or the other. Now the inclination of that evidence, such as it was, told rather for dynamic than structural disease; yet in about eighteen months later the patient came under my notice within a week or so of his death from one of the largest aneurismal sacs I have seen. *Per contra*, there is a case in the hospital books,[1] in which after very careful and repeated examination, the diagnosis of incipient aneurism of the vessel was set down in dubitative fashion, with a note of interrogation; and though the symptoms greatly improved under the rest and medical appliances of the hospital, they never did so in a sufficiently positive manner to induce me to modify the diagnosis into one of simple pulsation. Now this woman was killed by a street-accident two years later (in August, 1861); and her aorta, though somewhat thin and atheromatous, proved to be wholly free from dilatation.[2] I find it stated in my notes that at the close of the patient's stay under my care, the source of impulse was confined to a narrow surface at or near the median line.

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[1] Campbell, U. C. H., Females, vol. xv. p. 251.

[2] Davidson, U. C. H., Males, vol. xiii. p. 67.

[3] Brackenbury, U. C. H., Males, vol. v. p. 38, March, 1850—a case of this class. I saw this patient again in the autumn of 1853, he was then practically free from the complaint; but he came a third time under my notice, March 15, 1859, and had then been again suffering steadily from it for upwards of four years.

And there are occasional extra sources of perplexity. Thus, where an aorta, healthy in itself, is pushed forwards by an enlarged vertebra, or a tumor connected with the spine—or where an indurated mass lies in front of, or to either, or both, sides of the vessel, lateral expansion may be simulated, the murmur may be harsh, though not grating—while local pain and general emaciation may be produced by the organic morbid state, whatever it is, superadded to the pulsation.[3] Under these circumstances, the difficulty of diagnosis may be extreme. It appears from the case just referred to, that even the discovery of diastolic murmur in the vessel, that is, synchronous with its own systole, will not prove the existence of aneurism positively; still such murmur very rarely attends inorganic pulsation. As a rule, too, the murmur of the latter origin is inaudible in the back; but that of aneurism may be similarly limited in extent: the sex of the patient may give accidental aid; for aneurism of the abdominal aorta is very rare in the female, aortic pulsation common. The gravest examples of functional palpitation I have met with have, however, been in males. Again, the existence of inorganic murmur in the heart, thoracic aorta, and veins may assist also; but it may tend to deceive too, for the subject of aneurism is not exempted from becoming anæmic. In ordinary cases, then, the diagnosis is easy; in some rare instances it will be well to watch the case for a time before risking an opinion; and to positively affirm the absence or presence of slight peripheral dilatation of the coats of the vessel (Diag. III., fig. 1, p. 345), may, even after prolonged observation of the case, remain impossible.

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[1] Campbell, U. C. H., Females, vol. xv. p. 251, Sept. 1859.

[2] I am indebted for the opportunity of examining the aorta to the kindness of Mr. Miller, house-surgeon at the time.

[3] Lucas, U. C. H., Females, vol. xv. p. 312; here simple pulsation was transmitted some inches to the right of the median line through tumor.

A very remarkable case of combined aortic pulsation and movable left kidney once fell under my notice. The most interesting facts of the case were, that when the kidney lay, or was brought by the hand, in front of the throbbing vessel, the proper substance of the organ itself not only seemed to pulsate strongly, but with an action most distinctly expansile to the feel. I learned in truth, in a manner never to be forgotten, the danger of trusting to the seemingly "expansile" character of any given pulsating action, as distinctive of true aneurismal beat. The peculiarities of each of the two affections in this case threw light on the diagnosis of both; and there was no difficulty in forming an opinion of their nature.

808. *Prognosis.*—It is inferrible, from the foregoing account, that our promises must be cautious as to the duration or eventual curability of the complaint. But, though it may be sufficiently tormenting to embitter existence, I am not aware of its ever having destroyed, or even of its having ostensibly shortened, life.

809. *Treatment.*—In the majority of cases, the treatment of aortic pulsation directs itself to the removal of its causes—such as spanæmia, spinal irritation, gastric or intestinal disturbance, or leucorrhœa.

When the affection is more purely nervous, its management should be conducted as follows: all suspected articles of food, strong tea, and coffee, and similar stimulants should be forbidden, and a plain, nutritious diet rigidly adopted, with but little vegetable or other substances that promote flatulence. An occasional warm aperient, with some carminative adjunct, is advisable. Antispasmodic medicines afford relief to the symptom, though they fail to reach its cause; of these, valerian, æther, ammonia, assafœtida, and musk may be employed; sedatives, such as lettuce, conium, and hyoscyamus, may be given in alternation with others of the class acting more especially on the heart, namely aconite, digitalis, and hydrocyanic acid. These medicines are beneficially combined with tonics, where the general indications for such agents present themselves.

The application of some four or five leeches to the epigastrium sometimes distinctly tranquillizes the pulsating vessel—even where no fair suspicion exists of the presence of gastritis in any form.

Dry-cupping I have sometimes found beneficial, however difficult of explanation the fact may appear. Anodyne embrocations, belladonna plaster, and the endermic use of morphia in the scorbiculus cordis, moderate the arterial action.

Change of air and travel, moderate exercise, daily friction of the skin, the shower-bath, sea-bathing, or the tepid salt-water bath, and in fact, all hygienic influences, that strengthen the nervous system and improve the health generally, are among the most effectual agents in the management of obstinate cases.

§ II.—HYPERÆSTHESIA OF THE AORTA.

810. Hyperæsthesia of the aorta may exist in all degrees between downright neuralgia and mere unnatural consciousness of pulsation.

I believe that calcification of the vessel is sometimes the source of severe pain in its tunics, especially when the circulation is excited. Such pain has occasionally been confounded with that of genuine angina pectoris. It has occurred to me as highly probable that the peculiar pains felt in the limbs by certain phthisical patients, and which very decidedly do not follow the course of the nerves connected with the spinal system, may depend on excess and perversion of vaso-sensory innervation. They are sometimes spoken of by the sufferers as intermittently or remittently rhythmical.

Unnatural consciousness of the action of the vessel (and this may extend to a great portion of the arterial system), is a source of annoyance to some hysterical women. I have known the same symptom precisely complained of in anomalous states of the nervous system in the male.

§ III.—INFLAMMATION OF THE AORTA.

ACUTE AORTITIS.

811. Acute aortitis is a rare disease, at least as far as demonstration of its existence goes; it appears to be singularly uncommon, where it might be frequently expected, namely, in connection with acute endocarditis.

812. *Physical signs.*—The signs of this affection are obscure—at least in the present state of knowledge. Violent pulsation of the vessel, and tumultuous action of the heart, sometimes exist; it is very probable that, if lymph be deposited to any extent on the lining membrane, thrill may be perceived, where the vessel nears the surface of the chest. In a remarkable case, observed by Dr. Parkes,[1] an extremely loud, rough systolic murmur continued audible from the third dorsal vertebra quite down to the lumbar region—a murmur obviously due to the passage of the blood in the vessel over a surface roughened by patches of lymph. In this

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[1] Medical Times, Feb. 23, 1850.

instance the pulse was irregular and small, but the aortic orifice was contracted, and otherwise diseased, and the heart in a state of dilated hypertrophy; it does not appear that in the uncomplicated inflammation the pulse becomes irregular.

813. *Symptoms.*—Intense general uneasiness and jactitation are very usual symptoms; diffused tenderness of the skin has been noticed by Dr. Bright; rigors announce the onset of the disease; and M. Bizot insists much on the significance of general acute œdema of the trunk, arms, legs, and face. Dyspnœa, it appears, may be absent, when the disease is simple; yet it is difficult to conceive that the vessel can be inflamed to any extent without affecting the respiration: generally speaking, other affections, directly implicating the action of the lungs, coexist. From some observations by Dr. Corrigan, it would appear that inflammation of the mouth of the aorta may induce a series of pseudo-anginal symptoms; but on the other hand, that such is not a necessary effect is shown by Dr. Parkes's case. Pain, with sensation of heat, in the course of the vessel, complained of inferiorly, both anteriorly and posteriorly, on the level of the lumbar spine, has occasionally been a prominent feature. Syncopal tendency and apprehension of immediate death were noticed by Dr. Corrigan. Of the state of the urine nothing is known; yet, as we shall presently see, this is probably a matter of considerable importance.

Dr. Chevers, who has worthily labored on the diseases of the vessels, infers from collated cases, that death occurs in acute aortitis with extreme prostration, sharpened or bloated and livid features, cold and discolored surface, rapid indistinct pulse, stertorous respiration, swollen extremities, and duskiness of the superficial veins; the patients die comatose, and altogether with the aspect of persons destroyed by an animal poison. There is an asthenic variety of the disease, too, in which the symptoms are adynamic from the outset.

814. *Diagnosis.*—It will, I think, be generally conceded that the elements of a positive diagnosis of acute aortitis are yet to be found. If narratives may be implicitly trusted to, cases occur where, supervening on other affections, acute or chronic, acute aortitis produces no obvious effect except increased irritability and distress: clearly, the diagnosis of the disease could not, under such circumstances, be ventured on. Pain, thrill, and pulsation in the course of the vessel, with arterial murmur coasting the spine, and answering in localization neither to a murmur of the aortic nor of the mitral valves, would be the conditions, coupled with great general distress and pyrexia, most nearly warranting the diagnosis of the disease. But it is needless to point out the varieties of states that might simulate the entire series, except the aortic murmur; and in respect of this murmur, the possibility of its depending on chronic disease proclaims the necessity of caution.

According to M. Bizot, œdema of the trunk, arms, face, and lower extremities, occurring acutely, without functional disturbance of any organs but those of the circulation, indicates acute aortitis. But, on the one hand, I have seen the anatomical evidences of acute aortitis, where no anasarca had occurred;[1] and, in like manner Dr. Parkes's case, one of the most indubitable on record in regard of anatomical characters, proves absolutely that the disease may exist in perfection without any such œdema: while, on the other hand, M. Bizot has not taken into consideration the state of the kidneys in his patients. Now, in one of the three, Bright's disease appears to have been positively present; and in the other two we have no assurance that it was absent. M. Bizot's observations were made before renal diseases were either clinically or anatomically paid much attention to.

815. *Prognosis.*—Aortitis may prove very rapidly fatal; it has certainly destroyed life in three or four days in association with other less serious states. The transparence of exuded lymph furnishes the best measure of the recency of the disease. Of the prognosis, all that can be said is, that as persons die of various diseases with the evidences of chronic inflammation in the vessel, either acute aortitis sometimes fails to kill, or chronic aortitis sometimes pursues a chronic course from the outset; both propositions are probably occasionally true.

816. *Treatment.*—Were the disease upon fair grounds even suspected, active measures should without delay be had recourse to. Venesection or free cupping in the course of the vessel in the front of the chest and along the spine, or, if the disease have occurred in a very low state of system, dry-cupping, are clearly indicated. Counter-irritation, by the application of a long narrow blister along the left vertebral groove, which may be used also for the purpose of applying calomel and morphia endermically, has theoretical argument in its favor; for the mass of tissues, intervening between the skin and vessel, is in all probability sufficiently great to render the effect of the blister antagonistic.

817. The internal medicines, deserving of most confidence, are calomel, opium, and tartarized antimony: if the excitement of the heart and vessel were very great, digitalis or acetate of lead might be simultaneously administered. Aperients and saline diaphoretics are worthy of attention as adjuvants.

In employing these and other measures the practitioner must never lose sight of the constitutional state.

CHRONIC AORTITIS.

818. Thickening of the coats of the aorta, undue vascularity of the outer membrane, unevenness, roughness, puckering, furrowing, and channelling of the inner surface of the vessel, with various alterations in its calibre, producing irregular distension and con-

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[1] Campion, U. C. H., Females, vol. vi. p. 41.

traction, constitute unquestioned characters of chronic inflammation. White opaque cartilaginiform patches, studding the inner surface of the vessel, are likewise admitted to be the chronic representatives of the lymph-exudations of the acute disease. The relationship of saline precipitation to these patches is made matter of dispute; the fact appearing to be, that, while the usual nidus of calcification is certainly atheroma, the white patch is the occasional seat of the change, either primarily or secondarily to the deposit of atheroma within itself.[1]

819. But, serious as these conditions are anatomically, any symptoms to which they give rise, unless they have led to, or, at least, are attended by, very considerable alterations of calibre of the aorta, in the forms of obstruction, coarctation, or dilatation, prove, as a rule, obscure. For, although I cannot avoid mentioning, on clinical and *post-mortem* evidence, that marked calcification may prove the source of notable pain, yet, on the other hand, that such change of structure, even on an extensive scale, may remain wholly painless, is indubitable. And, again, until the elasticity of the vessel has been very deeply impaired, it continues capable of taking its natural part—in great measure a mechanical one—in carrying on the circulation.

§ IV.—ATHEROMA AND CALCIFICATION.

820. That atheroma and calcification of the aorta, no matter how interesting they may be anatomically, possess, *per se*, but a limited amount of symptomatic importance, flows directly from what has been said in the previous section. On the other hand, destroying, as they do, the elasticity of the vessel, rendering it fragile, contributing to, or at least facilitating, the production of various aneurismal changes, and connected with two important diatheses—the fatty and the calcifying—the prominence of these conditions of the vessel can scarcely be overrated in a pathological point of view. Besides, in their ultimate possible influence they are of grave clinical interest; for calcareous matter may be the occasion of obstruction, it may be obliteration, of the vessel (in its abdominal portion) by protruding more or less into the interior, and affording points for the blood to coagulate around.

821. Unless such mechanical results are produced, both subjective and objective evidences of the anatomical changes are wanting. Atheroma never causes pain that I know of; the occasional influence of calcification in this way has already been admitted. Neither condition necessarily interferes with the movement of the blood-stream.

822. And of physical signs of atheroma there are none concerning which I feel sure. But calcification of the arch is attended with systolic rough murmur, either limited to the vessel, which is

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[1] Products, Adventitious, Cyclopædia of Anat., p. 87.

rare, or audible also in a less intense form at the aortic valves—jerking, inelastic impulse behind the sternal notch, and, occasionally, systolic thrill both at that point and about the second right cartilage.[1]

§ V.—ANEURISM OF THE AORTA.

823. I. The term "aneurism," understood in its widest sense, may be defined as a local increase of calibre of an artery. And in this sense it has been used by some authors, while others have made attempts to restrict its application in many different ways. Professional opinion is, indeed, so unsettled as to the proper application of the term, that an explanation of the meaning he, in particular, may attach to it, is called for on the part of every person employing it.

824. Adhering, then, to the comprehensive definition above expressed, I would divide the *genus* Aneurism into the subjoined *species* and *varieties*:—

A. Peripheric: *dilating.*	Fusiform. Globular.
B. Lateral: *sacculating.*	a. Simple. b. Compound. c. Mixed.
C. Interstitial: *dissecting.*	

The anatomical constitution of these varieties of aneurism is exhibited to the eye in the subjoined diagram.

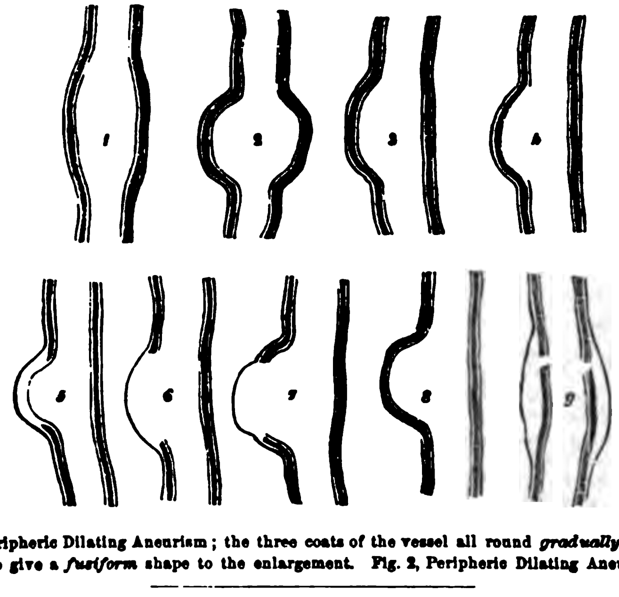

Fig. 1, Peripheric Dilating Aneurism; the three coats of the vessel all round *gradually* widening, so as to give a *fusiform* shape to the enlargement. Fig. 2, Peripheric Dilating Aneurism; the three coats of the vessel all round *abruptly* widening, so as to give a *globular* shape to the enlargement. Nos. 1 and 2 are the "simple dilatations" of various authors. Fig. 3, *Simple Lateral* Sacculating Aneurism; the sac, formed of the three coats of the vessel unbroken, rises from a limited portion of its circumference: the "*true* aneurism" of some authors. Figs. 4, 5, 6, 8, varieties of *Compound* Sacculating Aneurism; all agreeing in the characters of lateral sacculation, and injury to the coats of the vessel: the "false aneurism" of writers. Fig. 4, the sac formed of the middle and outer coats, the inner being destroyed. Fig. 5, the sac composed of the outer coat lined by the inner, the middle tunic having disappeared. Fig. 6, the sac composed of the outer membrane alone. Fig. 8, the sac composed of the middle and inner coats, forming a sort of hernial protrusion through the destroyed outer coat. Fig. 7, Mixed Aneurism, a combination of the simple and compound sacculating varieties; the simple condition of Fig. 3 having existed for a variable time, the middle and inner coats more or less suddenly give way, and the condition exhibited in Fig. 6 is added to the original simple disease. Fig. 9, Dissecting Aneurism; separation of the outer coat from the middle, by blood escaping from the interior of the vessel, through fissures in the lining and middle tunics.

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[1] Allen, U. C. H., Males, vol. xi. p. 330.

825. Now, in the aorta, the only vessel with which we have here to do, certain of the above varieties are distinguishable from each other by symptomatic characters, and, in reference to this important practical truth, may be placed in four *clinical* groups, thus:—

(*a*.) The simple and compound lateral sacculating aneurisms of Figs. 3, 4, and 6.

(*b*.) The fusiform and the globular dilating aneurisms of Figs. 1 and 2.[1]

(*c*.) The mixed aneurism of Fig. 7.

(*d*.) The dissecting aneurism of Fig. 9.[2]

By far the most important of these groups is the first. Its constituent varieties in the present state of knowledge are not distinguishable by any positive signs during life; hence their association in this clinical scheme.

826. The effects, signs, and characters of aneurisms of the aorta differ so much in the chief divisions of the vessel that the separate consideration of the disease, first, in the arch, with the ascending aorta conjoined, secondly, in the descending thoracic, and, thirdly, in the abdominal divisions, is matter of absolute necessity.

I.—ANEURISM OF THE ARCH OF THE AORTA.

827. GROUP *a*.—*Simple and Compound Lateral Sacculating Aneurisms.*—Affect a portion only of the circumference of the vessel; are generally narrower (the compound much more so than the simple) at the neck than at the body of the sac; rarely spring

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[1] Globular dilating aneurism, apparently most common in the cerebral arteries, I have never seen in the aorta until quite recently—May, 1852. The specimen is now placed in the U. C. Museum, No. 4036.

[2] I strongly doubt that the sac in Fig. 5 is such as it has been represented to be by authors—namely, the outer coat lined by the stretched inner membrane. The apparent inner tunic is probably nothing more than a film of adventitious formation; at all events, I have never seen a sac so constituted. Of Fig. 8 there is but one positive example known, so far as I can ascertain—namely, the preparation, No. 1642, in the Hunterian Museum. Another variety of hernial aneurism, protrusion of the inner through the middle and outer coats, has been described—in all probability, imaginatively.

from the inferior curvature of the arch; range in size from that of a nut to that of the fœtal head; and are filled to variable extents with laminated coagula—the compound more extensively, as a rule, than the simple, because the orifice of its sac is comparatively narrow. The influences of these aneurisms must seriously differ, according to the precise direction in which the sac chances to enlarge; but a grand practical distinction of specimens belonging to the present group may be established, in this point of view, into the centrifugal and the centripetal—those tending to grow outwards towards the walls of the chest, or inwards towards deeply-seated parts. The former habitually attain much greater bulk than the latter, for the simple reason that organs of vital importance being secure from pressure, the essential functions are, comparatively speaking, but little interfered with, life is prolonged, and time given for the free enlargement of the sac.

828. The effects of an aneurismal sac on the adjacent parts are of various kinds: but all, directly, or indirectly, due to pressure. Mere *displacement* is illustrated by detrusion of the trachea, or œsophagus, sidewards or backwards, of the heart downwards; by anterior bulging of the walls of the chest; by dislocation of the sternal end of the clavicle,[1] and by depression of the apex of the lung.[2] *Interference with the freedom of hollow canals* is seen in obstruction of the œsophagus, the trachea, either large bronchus, the descending cava, the innominate, left carotid, or left subclavian arteries—vessels which, blocked up either by direct pressure, by twisting of their orifices, or by coagula, become more or less functionally disabled. *Injury to parenchymata* is exhibited in compression of lung-substance, condensed so as to be incapable of expansion. *Destructive and perforative absorption of tissue* may occur in the trachea, the œsophagus, the pleura, lung, and bronchi; the pericardium; the pulmonary artery; the substance of the lung itself; the thoracic duct; the ribs,[3] sternum, rarely the clavicle, the vertebræ—though aneurism of the arch seldom, however, actually makes its way into the spinal canal; the recurrent nerve, and the spinal nerves on the left side. *Exudation of lymph* often takes place between the aneurismal sac and the parts pressed on, as the ribs and sternum, the pleura and lung. *Irritation of nerves* is exhibited in various spasmodic phenomena connected with traction of the recurrent nerve. And, lastly, actual *inflammation of texture*, as an indirect result of

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[1] The disarticulated head may sometimes be distinctly felt rising from its normal site; *e. g.*, Covey, U. C. H., Males, vol. xi. p. 316.

[2] Brader, U. C. H., Females, Clin. Lect. "Lancet," loc. cit., p. 117. No lung-substance reached higher in this instance, than the second rib on the affected side. The aneurism sprang from the descending portion of the arch, just at its union with the transverse, and was combined with small fibro-fatty tumor in both mediastina.

[3] Small aneurisms destroy contiguous bone more readily than large ones—obviously because pressure is more concentrated in the case of the former.

pressure, is displayed in the tracheitis and bronchitis that so often accompany the disease.[1]

Now, as will be readily understood, many of the different effects, just enumerated, are useful in guiding the observer to a precise localization of the disease. There is a peculiarity occasionally observed in connection with these effects, which, unless carefully interpreted, may lead to grave error of diagnosis. Some one or more of the number may disappear, giving place to others: thus, well-marked œdema of the shoulders and lateral base of the neck, caused by pressure on the corresponding innominate vein, may go wholly away, giving place to bronchial irritation. The explanation is simple: the direction of enlargement of the sac has changed in such manner as to remove pressure in one spot and transfer it to another. The same kind of change, though to a less degree, is obvious in occasional cases of mediastinal tumor; and in the instances of both aneurism and solid growth, I have known the diagnosis contested on the ground of such change.

829. *Physical signs.*—The physical signs of a sacculated aneurism of the arch are in well-marked cases extremely numerous, and as precisely and directly significant as, perhaps even more so than, in any other disease. But, on the other hand, the instances are rare, where the signs are numerously associated, unless the dilatation have attained very considerable dimensions; any one of the series of signs may be absent throughout the course of the disease; and sometimes during its earlier periods there is not a single one of the number satisfactorily developed. Hence it comes to pass, that the diagnosis of aneurism of the arch may be the easiest or the most difficult of achievements.

(*a*.) By inspection, local bulging is discovered, tending, when at all notably prominent, to the gently conical form. The ribs and interspaces equally contribute to its formation, when the area of the bulged surface is at all extensive; but when of limited extent, that is, at the time the sac commences first to act on the surface, a single rib or cartilage may alone be prominent, or the end of the clavicle simply pushed forwards. In cases of long standing, and where the base of the conical bulging is extensive, the skin becomes smooth and glazed-looking, the unevenness of the ribs and interspaces being completely removed. Concerning the seat of this bulging: if the sac spring from the first division of the arch including what is called the ascending aorta, the prominence mainly appears at the right edge of the sternum, in the second interspace and second cartilage, up to the first, and even as far sometimes as the clavicular joint. But if, while the sac involves this division of the arch, it likewise implicates the transverse and somewhat the descending portions,

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[1] For further illustrations of the effects of intrathoracic pressure, I must, in order to avoid repetition, beg to refer the reader to the section "Pressure-Signs" in the volume on the Lungs, p. 151.

the prominence, instead of lying to the right, may be placed solely to the left of the sternum, in the infra-clavicular and mammary regions, extending as far outwards as the line of the nipple;[1] obviously the ascending part of the arch gets twisted to the left. Secondly, if the transverse division be the affected part, the top of the sternum, or the right first cartilage forms the prominence; but if the sac be of small size, actually limited to this segment of the vessel, and springing from its posterior aspect, there may be, or, more correctly, will be, no visible bulging at all. Thirdly, when the descending portion of the arch is sacculated, the prominence appears at and about the second left cartilage: but aneurisms thus situated do not often cause anterior bulging; the vessel is here far away from the front of the chest, and, as matter of experience, the tendency of its aneurisms is to enlarge to the left side, while they expend their force posteriorly in eroding the vertebræ. Whatever be its seat, the superficial extent of the prominence is less than the diameter of the sac; the bulged surface corresponds to the most prominent part of the latter. The actual area varies between the size of half-a-crown and that of a large cocoa-nut. Deficiency of bulging is rarest where the sac lies to the right; commonest, where it lies to the left; of medium rarity, where the horizontal and central portion of the arch is implicated.

Movement, pulsatile, expansile,[2] and synchronous with the heart's systole, may be seen in the bulged part of the anterior chest; and, in some cases, motion of more abrupt and non-expansile character is very perceptible to the eye above the clavicles, evidently coming from below and as evidently produced by a body of some size. But if a sac, even though of massy bulk, be filled in great measure with fibrin, while a dwindled current of blood trickles with more or less difficulty through a small channel at the furthest aspect from the anterior surface of the chest, that sac may, especially if seated in the descending part of the arch, be as absolutely pulseless to the eye as if totally unconnected with the arterial system.

(*b.*) The hand, applied to the bulged surface, appreciates more accurately the motion of the sac—generally a very little behind the apex-beat of the heart in point of time, its systolic impulse is sometimes, as far as the sense of touch can determine, synchronous with this. By systolic impulse we mean that synchronous with the systole of the heart: if the vessel alone were considered, this impulse would of course be called diastolic. In certain instances, by no means in all, the impulse is double, a receding as well as an expanding motion existing—nay, in some cases of the sort, the diastolic action amounts to distinct impulse against the chest-wall, instead of, as usual, a mere receding jog. The force of the systolic impulse is sometimes extreme; even from a sac of small size, and mainly seated behind the sternum, which may have undergone but very slight erosion, the throb may be sufficiently strong to shake the head applied to the stethoscope.[1] I have known the diastolic impulse even similarly forcible.[2] In character, throbbing simply or thrusting and heaving, dull and inelastic (the latter condition marked in proportion to the quantity of fibrin in the sac), there are cases in which it is very positively undulatory. When the walls of the sac reach directly to the surface—when they have bulged this considerably—when there is a free passage through the sac—and when laminated coagula in but small quantity line its interior, the current of the blood passes with wave-like motion directly beneath the finger laid on the skin.[3] The seat of palpable is the same as that of visible action. When the descending portion of the arch is aneurismal, the impulse of its transverse division, which we will suppose sound, may be considerably intensified, as felt by a finger placed in the sternal notch; or it may vary from day to day, independently of any concomitant change in the impulse of the prominence directly over the sac—a circumstance sufficiently calculated to puzzle the observer as to the real seat of the disease. Aneurisms seated in this part of the arch tend also to raise its transverse division slightly above the natural level. In rare instances, very positive impulse may be detected in the inter-scapular region, more frequently on the left side than the right—a fact explained by the relationship of the arch on the two sides to the spinal column. Sometimes unnatural impulse in the region of the arch may be detected by laying one hand on the top of the sternum, the other in the inter-scapular region—the single hand having failed to distinguish anything abnormal.

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[1] Harris, U. C. H., Males, vol. ii. p. 262. The sac in this remarkable case commenced two inches above the aortic valves, and terminated close to the left subclavian; the innominate artery was carried to the left, so as to lie in front of the origin of the left carotid.

[2] The expansile character of the pulsation was shown to the eye remarkably in the case of Warren, U. C. H., vol. viii. p. 25, 1849. A belladonna plaster had been placed over the aneurismal bulging, which was remarkably broad-based; as the plaster produced an uncomfortable feeling of tension at first, it was slit across horizontally; subsequently, with each throb of the sac, the edges of the incision diverged by about a line.

Systolic vibratile thrill of variable amount may attend the impulse, or be perceptible even where distinct impulse cannot be detected. In aneurisms of the group under consideration, thrill is less constant than in the group of peripheric dilatations; but I have known it intensely marked, sufficiently almost to tickle the hand. It appears to attain its maximum in cases of lateral sacculation grafted on general dilatation. Thrill may be limited to the bulged surface, extend slightly in all directions beyond its confines, or be perceptible above the clavicles and sternal notch. The sign may be more distinct in the latter situation than over the sac itself. Thrill may exist at one time, disappear and return more forcibly than ever—a fact in some instances explicable by the varying state of the blood in respect of anæmia, in others referable to changes in the state of the sac, its contents, and its inlet.[1]

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[1] Downie, U. C. H., Males, vol. iv. p. 273.

[2] ———, attending U. C. H., 1861.

[3] Harris, U. C. H., Males, loc. cit.

The vocal fremitus may be annulled, over a sac of medium size, under the right clavicle.[2]

(*c.*) Aneurismal sacs, according to the direction in which they point, increase the measured distance from the sternal notch to the nipple, the middle line to the nipple, or both. They interfere with the measurable chest-expansion of breathing on the surface corresponding to themselves, and may restrain this very greatly by pressure on a chief bronchus.

(*d.*) Dulness, with resistance, under percussion, exists in the surface nearest an aneurismal sac. The reasons why the superficial extent of dulness should prove, practically speaking, less than that of the aneurismal pouch and artery, from which it springs, combined, have already been explained [261]. An area of some square inches may be rendered dull by sacs of very large dimensions. The observer must always bear in mind the possibility also of the real dulness of a sac being factitiously increased in extent by adjacent consolidated lung, or solid accumulation in the mediastinum.

The seat of dulness in *front* of the chest bears the same relationship, as that of prominence, to the different portions of the arch. As a rule, it is most easily and completely detected to the right of the sternum and in connection with the ascending portion of the arch and its angle on the right side; but if twisting of the vessel take place, the deficiency of resonance, even though the ascending portion of the arch be implicated, strikes us most obviously to the left of that bone.[3] Nor can it be said that a sac springing from the descending portion of the arch, even when small, fails to affect the percussion-sound; but, for obvious topographical reasons, it does so proportionately less, both in superficial extent and in amount. A sac originating in the horizontal part of the arch impairs the natural osteal quality of the resonance immediately below the sternal notch, the edges of the lungs being pushed sidewards to a variable extent: it also affects the sound above and to either side of the bone. Whatever be the seat of the pouch, it is important to ascertain positively whether the dulness connected with it does, or does not, reach completely into the acromial angle of the infra-clavicular region. Aneurisms of moderate size seated in the horizontal and descending parts of the arch give dull sound *posteriorly* in the inter-scapular regions; and a large sac, even though derived from its ascending portion, will impair the resonance between the right scapula and the spine.

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[1] *E. g.*, Case of Harris, loc. cit.—Thrill disappeared December 4, returned December 21.

[2] Mr. ——, seen with Mr. J. Tapson, October, 1852.

[3] Harris, U. C. H., Males, loc. cit.

If a sac be moderately stratified internally with fibrin, the character of its resistance is nowise special. If, on the contrary, it be closely filled with such coagula, and the amount of fluid blood within it be small, the resistance is dull, inelastic, and putty-like. This kind of resistance is significant enough, when discovered; but it is scarcely necessary to add, that either for the purpose of eliciting this, or any other, character of percussion, the least roughness in manipulation is not only awkward and *unclinical*, but actually dangerous.

In a former place [261], I have touched on the question of the smallest amount of dilatation of the arch capable of being demonstrated by percussion. Under a concourse of favoring circumstances, we have seen that a very small increase of size may be so discovered. The inefficiency of percussion increases with the smallness of the sac and its distance to the left of the median-line. There can be little doubt that more than one small aneurismal sac, in the middle division of the arch, has escaped detection, simply because percussion was not performed below, and at, the sternal notch.

(*e.*) Few diseased states give rise to such variable auscultatory signs, as a sacculated aneurism of the arch—a fact sufficiently proved by the following series of conditions of sound that have actually fallen under my notice: the list probably might be increased from the experience of others. Through the stethoscope, placed on the most prominent part of the surface, I have heard—

1. A double sound, both divisions of which are rendered murmur-like by suspension of the respiration, and both are weaker than the sounds at the base of the heart. 2. A double sound of the same characters, except that its divisions are louder than the basic sounds of the heart. 3. No sound at all, properly speaking; but a dull, impulsive impression, systolic in time, that simulates sound. 4. A systolic impulsive impression of the kind, with a pure ringing second sound.[1] 5. A systolic blowing murmur, harsh in quality—harsh to the same, a greater, or a less amount than elsewhere in the artery close to the sac; with a dull muffled diastolic sound. 6. A roaring, grating systolic murmur, stronger than at the base of the heart, the aortic valves being constrictively diseased, and the blood spanæmic; with a diastolic sound. 7. A double rough murmur, the systolic division louder than the diastolic. 8. A double rough murmur, the diastolic division louder than the systolic. 9. A systolic sound, with a diastolic murmur—a very rare combination. 10. In systolic time, sound and murmur both, and distinct from each other; in diastolic time, a loud, ringing, clanging sound.[2]

It is to be understood of all these varieties of aneurismal audible signs, that the influence of valvular disease in their production is, as matter of observation, supposed to be excluded.

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[1] Bell, U. C. H., Males, vol. vii. p. 168.

[2] Moriarty, U. C. H., Males, vol. xi. p. 164.

Aneurismal sacs may then be, as it were, silent, or be the seats of sound or murmur, either singly or both combined. On the relative frequency of these different acoustic states I can make no positive assertion; nor do the elements for such assertion exist anywhere, that I am aware of. But of one fact, that murmur is vastly less frequent than the language of the majority of writers would indicate, no one, accustomed to clinical observation, entertains the least doubt. Another fact not generally known, or at least habitually lost sight of, is that the conditions of sound or murmur over a sac not only vary at different periods of the disease, but even occasionally, from day to day, in different postures, and at different phases of the respiratory act.

The *sounds* heard over aneurismal sacs are, generally speaking, identical in special character with those of healthy arteries—they are simply more intense. But occasionally, a peculiar character in the single or double sound of an aneurismal sac, when completely free from murmurish quality, may be perceived, that seems best designated by the phrases *pumping* or *sucking:* this variety is not without diagnostic significance.

Of the particular attributes of aneurismal *murmurs*, the following are worthy of clinical attention. The quality of the *systolic* murmur may be simply blowing, or blowing with a peculiar hoarse hollowness (which, when well marked, is important in diagnosis), grating, rasping, sawing, filing, or, if the blood be spanæmic, roaring. Variable, but generally low, in pitch, this murmur may be of higher pitch, nevertheless, than a coexistent systolic murmur at the base of the heart.[1] Short and abrupt in the majority, prolonged, almost drawling, in the minority of cases, its intensity may reach, or even exceed, that of the loudest murmurs produced within the heart. The strength of the *diastolic* murmur, though variable, is rarely great, absolutely speaking: in quality it is generally softer than the systolic; it is not constantly present, not only at different periods of the same case, that has at one time presented it, but even with successive beats of the heart. Generally best audible over the most projecting point of the prominence, murmurs may, on the contrary, attain their maximum loudness at the edges of this: or, even when the main part of the aneurism lies considerably below the clavicles, they may be strongest at the base of the neck—probably, under the latter circumstances, thick layers of fibrin are accumulated in the lower parts of the sac: or, lastly, they may, in very rare cases, exhibit greatest intensity on the left side of the spinal column.

The mechanism of these murmurs seems to be as follows. The systolic is either produced by the passage of blood over a surface roughened by fibrinous masses, inequalities in the coats of the vessel, and calcifications; or it may come of the rippling motion given to the fluid on its entry from a portion of tube of natural calibre into one more or less dilated, especially if the dilatation be abrupt; or it may be caused by the flow of blood through the comparatively narrow and more or less rough orifice of the sac; or, not directly dependent on the aneurism itself, it may proceed from a spot of the vessel pressed upon and rendered narrow by the sac. Or it may originate beyond the sac, and arise from the passage of the blood-stream from the dilated part of the vessel into that of natural calibre.[1] On the other hand, diastolic murmur seems due to the reflux of blood from the sac through its orifice; and as the force of the reflux current must be comparatively slight, so, as a rule, diastolic aneurismal murmurs are feeble: sometimes, where undue force is given to the back-current by coexistent aortic regurgitation, the diastolic murmur, especially if the sac be very close to the origin of the aorta, acquires unusual intensity. When the entry of the blood into the sac is murmurless, and its escape productive of murmur—that is, when diastolic murmur alone can be heard—the peculiarity probably depends on some special condition of the orifice of the sac, whereby a smooth surface is presented to the entering blood, and a surface, roughened by movable fibrin or otherwise, opposed to the receding current.

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[1] I do not believe, however, that an aneurismal murmur is ever of, absolutely speaking, very high pitch.

A number of conditions tend either to enfeeble or intensify aneurismal murmurs. If the heart's action be very weak; if the sac be filled in great measure with fibrin; if the sac be incapable of much expansion; if the orifice of communication between the sac and the artery be very large, inasmuch as the current is then too free; or if that opening be at once very narrow and smooth, inasmuch as possibly the stream is then too small to generate notable sound—if any one of these conditions be present, a sac, otherwise well constituted for the purpose, may fail to furnish murmur. On the other hand, great roughness of surface, neighboring pressure, and the presence of good conducting material round the sac, will intensify, really or apparently, these murmurs. Mere sounds may sometimes be rendered murmurish by suspension of the breath for a moment—a fact to which it is not easy to supply a satisfactory clue.

A murmur may be suddenly generated in a person, known subsequently to be the subject of aneurism of the arch, be audible not only to himself, but to bystanders at some distance off, pervade the entire arterial system, though of maximum loudness at the upper part of the chest, and disappear, at least as a phenomenon audible without the aid of auscultation, mediate or immediate, as suddenly as it came.[2] More than one hypothesis readily suggests itself in explanation of this singular condition; but, as I have no post-mortem evidence, proving the reality of any one possible mechanism rather than of another, I abstain from conjecture.

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[1] Mr. ——, seen with Mr. P. Duffy.

[2] Facts all illustrated by the case of Mr. ——, seen with Mr. Pollard, of Brompton: death occurred by rupture of the sac.

830. *Symptoms.*—(*a.*) The weight and flesh of aneurismal patients undergo very considerable reduction in a certain share of cases; while instances, where the sufferer continues stout to the last, or almost to the last, are far from very uncommon. The centripetal or centrifugal process of the aneurism does not always explain this difference; if, on the one hand, the greater disturbance of important functions, attached to the former mode of progress, tends to produce rapid emaciation, the protracted, though less acute, functional mischiefs endured in the latter eventually work out the same result. Besides, these two topographical modes of progress are sometimes so mixed up that in not a few cases it is impossible to place the disease exclusively in one or the other category. But from the analysis of seventeen cases of aneurism of the arch, of which I have notes, the inference clearly flows that the presence or absence of pain of serious character is the real element in determining, or warding off, early emaciation. And that pain is very decidedly greater in cases where the dilated vessel bears either against the chest-wall and intercostal nerves, or the spine and vertebral groove —hence where the course is centrifugal. It appears that extreme emaciation has sometimes been mechanically caused by pressure on the thoracic duct. The face, trunk, and limbs waste *pari passu*— the face not presenting that relative fulness often so remarkable in the emaciation of phthisis.

There is not any attitude, posture, or mode of decumbency peculiar to the subjects of aneurism of the arch, as a class and for a permanency; in bed the patient usually lies on the back, with the head moderately high. But for the relief of particular kinds of pressure, peculiar attitudes may be assumed; thus, where a sac bears upon the trachea, the patient steadily keeps the head forwards, or forwards and sidewards—and also frequently raises or throws back the head suddenly, keeping it in this posture for a time, so as to project the sac forwards from the windpipe.[1] When paroxysmal attacks of dyspnœa, from tracheal pressure or irritation of the recurrent nerve, occur, the sufferer sits up with his head supported on his hands, the elbows resting on the knees, or bends over the back of a chair, &c. The sleep is not affected by the aneurism itself; but if pressure exist, the ordinary slumbers are fitful, interrupted by starts, and frightful dreams. During the urgency of bronchitic and asthmatic seizures, the patient may pass night after night out of bed. The easiest attitude in sleep may be one which to a healthy person would prove unbearable: thus with the head supported sideways wholly on the hand;[1] postures of the kind may also be assumed consensually during sleep.

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[1] In a case recently seen, the existence of this habit, combined with slight gnawing interscapular pain, drew my attention to the arch of the aorta, where, with much difficulty, the physical signs of a small sac were elicited.

The expression of the face varies: it may be calm and not indicative of suffering, except during paroxysms of dyspnœa, growing then, terrified and imploring; or habitually cross and irritable; or anxious and worn; or simply significant of profound distress. The differences in the original temper of patients modify their facial expression during this, as all other chronic diseases. The color of the face may be to the last florid in the main, with slight lividity;[2] or habitually livid; or in no single point remarkable; or pale, sallow, and cachectic-looking: the latter alone is in the least degree distinctive. I have known the expression of the face singularly altered by distension of the angular and adjacent part of the frontal veins.[3]

(*b.*) The skin of the general surface, though sometimes sallow, cannot be said to exhibit any habitual special tint. Sweating, the reverse of an ordinary symptom, I have nevertheless known give considerable annoyance—and this where the lungs were sound. The lower extremities remain singularly free from œdema; a fact often finding its diagnostic application. I have known slight bulbousness of the finger-ends, confined to the side on which the supply of blood was limited through the influence of the sac.[4] Œdema of the base of the neck, the face, and the upper extremities, and one or both sides of the thorax, follows pressure on the superior cava, or one or both innominate veins. The peculiar spongy elastic fulness of the base of the neck, looking like a *collar* of flesh, due to capillary turgescence, is also observed. The integuments over the sac sometimes alone become œdematous, from irritation or mere distending pressure. The temperature of the arm, of which the circulation is impeded, may fall very notably below that of its fellow.[4]

(*c.*) The joints are not affected; no positive connection exists between either rheumatism or gout and aneurism.

(*d.*) The lips full, tumid, and livid, the tongue œdematous at the edges, and of purplish tint, the mucous membrane of the pharynx, thick, livid, and coated with viscid secretion, when other signs of venous pressure exist, display, if these be absent, no peculiarity of appearance. Dysphagia, slight or severe, felt more in some postures than in others, paroxysmal or permanent, or both combined, or in some severe cases disappearing completely for a while, after having been a constant condition (a change sometimes explicable by diminution of pressure through hemorrhage from the sac), is a symptom of considerable frequency. More commonly attending the disease in the descending and transverse parts of the arch, dysphagia may be absent even in the former case,[1] and coexist with a medium-sized sac springing from the right angle of the arch.[2] The intensity of dysphagia obviously depends in the main on the general and local nervous susceptibility of the patient; a slight amount of pressure on the œsophagus will produce greater difficulty of deglutition in some persons, than actual perforative destruction of the coats of the tube in another.[3] Blood may be discharged in large quantities, from rupture of the sac, into the œsophagus—an event which, moreover, need not prove immediately fatal. Whether slight oozing of blood may take place by filtration into the œsophagus, and be discharged by sputation, without actual hemorrhage, I do not know from experience; but there can be no reasonable doubt of the possibility of the occurrence. The appetite fails altogether, if the aneurism be the source of pain; commonly it is capricious. Blood in small quantities, darkened and otherwise altered by the gastric fluids, is occasionally vomited, after having trickled into the stomach from filtration through the walls of the sac and œsophagus; blood of similar origin may also be traced in the stools.[4] The bowels are habitually constipated in advanced cases, probably from the patient's inability to take exercise; flatulence distresses many, even male, patients; piles and pruritus about the anus seem to be more frequent than in the average of persons of equal age. Ascites does not occur.

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[1] King, U. C. H., Males, vol. vii. p. 89.

[2] Brader, U. C. H., Females, loc. cit.

[3] Moriarty, U. C. H., Males, vol. xii. p. 3.

[4] Mack, U. C. H., Males, vol. ix. p. 210

(*e*.) Painful, hoarse, clanging, laryngeal cough, laryngeal rhonchi, dry or moist, audible sometimes at a distance, and dyspnœa, at once habitual and increasing paroxysmally, coupled with various morbid states of voice, indicate deep disturbance, functional or organic, or both, of the larynx. The speaking voice may be husky, muffled, cracked, and hoarse; or simply weakened, or tremulous and variable in pitch, or actually lowered in register. The hoarse variety appears to depend on chronic laryngitis, with diminished current of air—itself, in turn, traceable to pressure on the trachea; pressure on a main bronchus is not sufficient for the purpose. Œdema of the glottis, depending on congestion, venous or sub-inflammatory, has in some cases been found. Paralysis and atrophy of the muscles of one side of the larynx, coupled with flattening and compression of the recurrent nerve, explained extreme vocal feebleness in a case observed by Dr. Todd. Tremulousness and variation of note have been traced to simple pressure on, and displacement of the trachea. Paroxysmal dysphonia is explained by irritative traction of the recurrent nerve. The trachea, chronically inflamed, where the subject of irritative compression, is tender to the touch; and stridulous breathing is in part due to these conditions.

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[1] Brader, U. C. H., Females, loc. cit.

[2] Downie, U. C. H., Males, loc. cit.

[3] A perforation the size of a shilling may exist without the very least dysphagia occurring during life; though, too, the current of blood in the sac must have borne directly almost against the gullet; though, too, the wall of the sac may be gone at the spot, and portions of its fibrinous coagula protrude slightly into the interior of the œsophagus: Brader, U. C. H., Females, loc. cit.; I repeatedly saw this woman swallow with perfect ease. In King, too, U. C. H., Males, loc. cit., a smaller perforation existed without dysphagia.

[4] I have met with one case of repeated hemorrhage through the mouth, having all the characters of hæmatemesis, and in which, the lungs and stomach not being demonstrably unsound, there were several signs and symptoms of aneurism of the aortic arch.

Various forms of pain are, or may be, felt in the chest. First, immediately over the aneurismal prominence, pain may be produced by mere distension, by local pleurisy, or by irritation of the intercostal nerves. Secondly, pain in the neck and arms, down to the finger-ends, is traceable to irritation of the branches of the cervical and brachial plexuses. Thirdly, pain of a peculiar gnawing, terebrating character, constant, but increasing paroxysmally from time to time, exists at the dorsal spine, if the vertebræ are undergoing absorption. Fourthly, local pain and tenderness over the sternum, is sometimes connected with periosteitis advancing to suppuration.[1] The second class of pains are of shooting, piercing, or stinging character, may be brought on by the slightest movement, even that of turning in bed, and are more or less paroxysmal. The gnawing pain in the back cannot, as some persons have supposed, depend on irritation of the roots of the spinal nerves, or it would, contrary to what is the fact, radiate in the course of their branches. The tenderness of an aneurismal prominence is sometimes extreme —and in certain cases associated with a sensation of heat, perceptible also to the hand of the observer. In addition to all these sufferings, a feeling of fulness, weight, load, tightness, and oppression is experienced within the chest, in some instances coupled with a dread of movement, lest something should be displaced by the change of posture. There are patients who suffer seriously from spasmodic contractions of the diaphragm, or sensation of constriction round the base of the chest—the obvious results of irritation of the phrenic nerve.

The state of the respiration varies. If there be no pressure on the tubes, or irritation of these, or pressure or irritation of the vagi, recurrent, or pulmonary nerves, the breathing is calm. Where any of these conditions are present paroxysmally, the breathing undergoes temporary acceleration in proportion to their amount. Or, if they are permanent evils, the breathing is labored, whistling, stridulous, audible at a distance, and the patient commonly points to the trachea, at the sternal notch, as the source of difficulty. The number of respirations per minute almost always exceeds the average of health more or less—I have found it range from twenty-four to fifty-six. The pulse-respiration ratio is subject to great variation; thus, in one of the cases already referred to, the mean ratio throughout the time of observation being as 2.9 : 1, the extremes were as 5.3 : 1, and as 2.3 : 1; in this instance, the variations of broncholaryngeal symptoms furnished a key to the rises and falls.[1] The chest-play is more or less confined. If a main bronchus be encroached on, or the mass of one lung diminished by pressure, the play on the corresponding side will be relatively deficient; and there may be special want of expiratory rather than of inspiratory power.[2] Dyspnœa in an almost invariable effect where the sac is of any bulk, though all annoyance of the kind may positively be wanting where the sac is even huge.[3] In many instances the first symptom attracting the patient's attention, and commonly increasing gradually in intensity, dyspnœa acquires, *cæteris paribus*, most intensity when the horizontal part of the arch is aneurismal. In addition to the pulmonary causes of the symptoms enumerated above, may be mentioned pressure on the auricles, pulmonary veins, or pulmonary artery, and passing accumulation of blood in the right cavities of the heart. Dyspnœa is prone, too, to occur paroxysmally at night, from accumulation of sputa, pressure on the trachea, produced by accidental movement into such postures as throw the sac against that tube, and probably from reflex action. The act of deglutition sometimes induces a severe fit of obstructed breathing.

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[1] Downie, U. C. H., Males, loc. cit.

Cough, rarely absent, may be loud, dry, and paroxysmal, paroxysms of the kind sometimes terminate in a syncopal state, or, after great effort, are relieved by expectoration of a thin, watery fluid. Unless, under the influence of accidental inflammation of the air-tubes, there may be no expectoration at all. Blood may be discharged through the trachea in different manners. The sac and windpipe undergoing an extensive rent, a tremendous flow of blood may take place, and kill instantaneously; or, syncope occuring after copious discharge, coagula form and plug up the opening for the time; upwards of a quart of blood may be poured out under these circumstances without immediately fatal result; still such temporary preservation of life is a rare exception to the common issue of such ruptures. Or, lastly, the expectoration may be habitually

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[1] Brader, U. C. H., Females, loc. cit. But this explanation will not always hold good; a ratio of 4.4 : 1 may coincide with very considerable irritation of the whole broncho-tracheal tract.—Mack, U. C. H., Males, vol. ix. p. 210.

[2] Thus, in Brader, the measurements of the chest below the fold of the mamma, gave,

	Medium.	Inspiration.	Expiration.
Right	17⅛	17¼	16⅞
Left	16½	16¾	16½

Hence the total play on the right side equalled three-eighths, on the left two-eighths, of an inch. The left bronchus was "distorted, and almost obliterated by pressure of the sac." Hence it appears, too, that the excess of the right over the left side, at the end of full inspiration, was half an inch; at the end of full expiration, three-eighths of an inch. These results prove that there was a deficiency of expiration on the left side. They are corroborated by the facts, that the difference between full inspiration and full expiration on the right side equalled three-eighths of an inch, on the left only a quarter of an inch; while on the left side there was no difference, on the right a difference of a quarter of an inch, between the medium state and full expiration.

[3] Harris, U. C. H., Males, loc. cit.

tinged with blood, so as to produce the red-currant jelly appearance; such expectoration, which is very unusual, does not always derive this character from blood filtrating from the sac, but sometimes from pressure on the vessels of the lung.[1] Moderate discharge of blood, by diminishing the size of the sac, and also by diminishing congestion, sometimes affords great temporary relief of urgent symptoms. The following brief abstract of a case which fell under my observation, some while since, illustrates some remarkable points in the natural history of aneurismal hemorrhage.

Mr. R. P., June 9, 1857. Ætatis about 45, complains of obstinate pains about right chest, with a feeling as if crushed on level of right pectoralis major, this sensation often waking him up at night. Nothing physically wrong in lungs; over-action of left ventricle, with toneless first sound; examination elicits no evidence of aneurism, carefully looked for on account of above symptoms. Dec. 7, 1857. Percussion-signs of aneurism, right second cartilage, the dulness extending across top of sternum; very slight conical-looking prominence in former site; pulsation faintly visible and palpable; no thrill; over prominence double sound, second twanging, no murmur; slight dysphagia; frequent giddiness; right radial pulse scarcely equals one-third of the left in breadth and force. January 4, 1858. The prominence has enormously increased; chest-wall feels so thin that dare not place stethoscope on it; radial pulses as before; right pupil slightly larger and more sluggish than left; occasional oppression; very slight cough; dysphagia as before. Rupture of the sac through the chest-wall now appearing imminent, I made arrangements with Dr. Allchin, who lived in the neighborhood of the patient, for such measures as appeared advisable in the event of its occurring. January 26, 11.30 P. M., rupture took place; 30 to 40 oz. of blood lost, without syncope; gradually ceased in about three hours. Gradual rally, with occasional oozings, till Feb. 5th, when copious flow occurred, though less than the first. Feb. 9, discharge to extent of about 40 oz. From this time to March 8th some five or six bleedings occurred, varying in quantity from 2 oz. and 3 oz. to 6 oz. or 8 oz.; on this day a serious discharge to amount of from 30 oz. to 40 oz.; from this to the 17th March frequent slight oozy discharges took place, on which day a sudden burst of blood was followed by instantaneous death, the clots resembling slightly colored size, and the discolored linen of salmon tint. During all this time the first local application was never removed, consisting of lint, tow, and matico-leaves, saturated with alum and tannic acid (the two latter frequently renewed); ice-bags were kept constantly applied also. From Feb. 5th to the end of that month the nervous erethism of the patient was so extreme as to render him at times almost maniacal; this state was controlled by the free use of morphia.

On removing the mass of lint, &c., after death, it was found this lay in direct contact in two places with coagula within the sac, the openings in the chest-wall being severally about the size of a penny and a halfpenny. Two fingers introduced easily reached the aortic valves. Hence this patient must have lived for nearly two months with a gradually increasing extent of his chest-wall and aorta replaced by lint.

Certain physical signs connected with the lungs are worth attention. Where the trachea is pressed on, the supra-sternal region sinks in very deeply on inspiration; rhoncho-respiratory fremitus, in consequence of the powerful stridor of respiration, is carried to its maximum point. Pulmonary percussion-dulness comes of various influences exercised by the sac on the substance of the lung, displacement, condensation by pressure, and collapse from obliteration of bronchial tubes. Respiration-sound may be deficient to almost suppression, from bronchial pressure, through part or the whole of one lung, while exaggerated respiration exists elsewhere. Condensed strata of lung yield high-pitched bronchial respiration; and dry and moist bronchial rhonchi are audible. Jerking rhythm may sometimes be caused by the pulsation of a loose sac against the trachea or lung.

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[1] Brader's case, loc. cit., p. 121.

(*f*.) The *heart* may become hypertrophous, especially if the sac originate near the sigmoid valves; but such effect is by no means constant; I have known its size fall quite within the limits of healthy bulk under the circumstances. In some cases, the relative widths of the arterial outlets of the heart undergo perversion; in one remarkable instance, the pulmonary orifice, when opened out, measured two and a quarter inches, while the aortic reached four and three quarters; this state of things must, in all probability, seriously increase dyspnœa.[1] Inequality of force and fulness of the radial, carotid, or subclavian pulses, at corresponding points of the two sides, occasionally exists. Though other explanations have been suggested, it appears that pressure or obstruction with coagula at the aortic origin of the weakly-beating vessel is the only positive cause of the difference.[2] The vessel on the affected, beats a little later, too, than on the sound, side. There is a certain sharpness and jerking character in the pulse, sometimes not unlike that of slight aortic regurgitation; but the superficial arteries do not beat visibly. The pulse is sometimes bisferiens; however, observation does not justify the notion that the second wave depends on reaction of the aneurismal sac. The veins of the chest, of one or of both upper limbs may be enlarged, full, and knotty, sometimes so firm that they cannot be flattened by pressure; those of the arm have been known to undergo complete obliteration by coagula.[3] I have never seen pulsation of the jugulars in these cases. The situation of the obstructed veins will guide to that of pressure, whether on the superior cava or either innominate vein alone.

(*g*.) The bronchial glands are sometimes enlarged, and increase the percussion-dulness of the aneurism in the back.

(*h*.) The urine frequently contains excess of urea; it is free from albumen: on the whole, it is, *quoad* diagnosis, insignificant.

(*i*.) The genital organs present nothing special.

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[1] Downie, U. C. H., Males, loc. cit.

[2] Change in the pulses may, as might be anticipated, be pretty speedily effected. Thus, in Mack, U. C. H., loc. cit., p. 210, "the left radial was feeble, yet sharpish, the right so excessively feeble that it was with difficulty counted." Dr. Reynolds had seen this patient one fortnight before, and was positive no difference then existed between the two pulses. Where difference of the kind occurs, the arteries of the lower extremities should always be examined, lest a normal irregularity in the two sides be taken for the result of disease.

[3] Ferrus, Mém. Acad. Roy. de Médecine.

(*k.*) Cephalalgia, a frequent symptom, sometimes depends on the throbbing action of the arteries, and is sometimes simulated by pain in the nerves of the scalp, from pressure on the plexuses below. Partial paralysis, sensory and motor, of an arm has occurred from pressure on the brachial plexus. The intellect is habitually unaffected to the last. It is quite conceivable that obstruction of the innominate or of the left carotid may be carried to such a point as to interfere with the nutrition of the brain and induce symptoms; but such result is, clinically, at least very rare. I have seen the available opening of the innominate reduced to the size of a crow's quill flattened, through combined twisting of the trunk and the obstruction of coagula, without any cerebral symptoms having occurred during life. Probably this absence of suffering on the part of the brain results from the slowness with which the obstruction is habitually worked out. For there is considerable reason to believe that when abruptly effected, as by dissecting aneurism of the arch, grave encephalic mischief may ensue.

(*l.*) Paraplegia has, in rare instances, followed erosive destruction of the vertebræ and pressure on the spinal cord. In one remarkable case, death occurring with sudden paraplegic symptoms and indications of rupture into the chest, the sac, it was discovered, had burst into the pleura, and also into the spinal canal.

(*m.*) As a rule, the organs of sense escape—at least, they escape such amount of implication as readily attracts attention. I have, however, known deafness supervene, when some form of connection with the aneurismal disease seemed highly probable.[1] It has not occurred to me to observe positive failure of vision in cases of aneurism of the arch; but I have met with four examples of inequality of the pupils since I first noticed the phenomenon, nine years ago.[2] These cases have shown me that when the pupils are affected, contraction on the aneurismal side may exist persistently; or the inequality may only be shown by sluggishness under light on that side;[3] or they may be variable, even within a few days—that on the aneurismal side being now equal to, now notably, now slightly smaller than, now larger than, the other in size.[4] The latter conditions would prove that the pressure of the sac must act now paralytically, now irritatively, on the sympathetic at the root of the neck.[1]

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[1] Moriarty, U. C. H., Males, loc. cit.

[2] Mack, U. C. H., Males, vol. ix. pp. 211, 244. "April 23, 1853. Left pupil, in medium state, about $\frac{1}{5}$ inch in diameter; right not more than half the size; both are round and moderately brisk." This state held on during life, but after death —"May 24, . . . pupils round, both larger than during life; the right, that which was during life so notably the smaller, is now very distinctly the larger, of the two." In this case the innominate artery was so compressed that a narrow button-hole slit alone opened into the arch, and the right radial pulse was almost imperceptible; but the relative state of the carotid supplies cannot be appealed to in explanation of the unequal sizes of the pupils—as Dr. Reynolds has seen them thus unequal at a time when he found the radial pulses equal.

[3] Hobson, U. C. H., Males, vol. xvi. p. 77, March 10, 1859.

[4] Covey, U. C. H., Males, vol. xi. pp. 318, 352; vol. xii. p. 31, 1855.

I have recently observed a case in which, along with contracted pupil, the temperature of the same side of the face was distinctly higher to the hand than on the other. Not having had a fitting thermometer by me, and having seen the patient but once, I am unable to fix the precise amount of difference. But the fact illustrates the results of M. Bernard on the section of the sympathetic in the neck.

(*n*.) In addition to the neuralgic pains in various nerves from pressure, absorption of their substance is sometimes effected. Intercostal neuritis has in a few instances been detected.

831. *Course.*—In the great majority of cases the progress of aneurism is slow and insidious at first: the symptoms may, however, be suddenly developed, probably from some sudden increase in the bulk of the sac—an increase generally connected with change from the simple to the mixed condition of the disease. The further course of cases is either gradually or interruptedly progressive: occasionally aneurisms of considerable dimensions have remained latent to the last, and their physical signs been merely those of a soft, solid mass within the chest. Physically considered, the course may be centripetal or centrifugal, or both combined: when aneurism combines both modes of progress, one almost always predominates in activity over the other.

832. *Mode of Death.*—There are five chief ways in which life may be destroyed in the subject of aneurism of the arch: by gradual asthenia and exhaustion; by the obstructive and irritative effects of pressure; by rupture and hemorrhage; by acute intercurrent diseases; by coexisting chronic diseases.

(a.) On the whole, actual death by gradual exhaustion is rare. Pain, insomnia, anorexia, wasting and low irritative fever, not unfrequently, it is true, bring the patient to the brink of the grave; but in several instances of the kind, observed by myself, the immediate cause of dissolution belonged to one of the other categories, especially rupture. In one melancholy case, which I saw some three or four times in consultation, the patient, unable to bear the anguish of intercostal and other neuralgic pains, which were fast undermining him, committed suicide [836].

(b.) Whether the aneurism affect the ascending or the transverse portions of the arch, broncho-tracheal pressure and irritation rank among the most frequent causes of death.

(c.) Solution of continuity in the walls of the sac may be effected by slow perforation, or sudden rupture. Perforation, with or without oozing, or slight pouring out of blood, may occur into parts of considerable importance, not only without immediately fatal results, but without any perceptible special effect;[1] the occurrence of a sudden rent of any size completely through the wall of the sac, no matter with what internal part an unnatural communication be thus set up, is however almost invariably fatal at once. A very large breakage of the adherent sac and chest-surface may however, as we have a moment since seen, take place with actual passing comfort from the ensuing hemorrhage. There is no conceivable position into which fatal rupture has not occurred, as may be seen by examination of Dr. Crisp's valuable collection of recorded cases. Where the ascending part of the arch has been affected, the pericardium has proved the most frequent seat of hemorrhagic effusion. Of 138 cases, where Dr. Crisp found the mode of death stated, six only furnished examples of external rupture.

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[1] Petit noticed in 1727, not only contraction of the pupil, but also increased vascularity of the side of the face on section of the sympathetic trunk in the neck. *Vide* Gairdner, Assoc. Med. Journal, 1855, and Ogle, Med.-Chir. Trans., vol. xli., 1860.

Hemorrhagic effusion from a sac may be retained, as when it pours into the pericardium, pleura, mediastina, spinal canal, pulmonary artery, vena cava, or various parts of the heart; it is rejected, when poured into the trachea, a bronchus, the lung-substance, the œsophagus, or through the chest-wall. Large amounts of blood may be discharged, not only without deleterious effect, but actually with beneficial influence, through relief of congestion, and diminution of various irritative pressures.

Death may also take place by rupture of the heart, or of the aorta itself on the cardiac side of the aneurism.

(d.) Death by *dependent* acute intercurrent diseases, such as pneumonia, gangrene of the lung, bronchitis, pleurisy, pericarditis, is occasionally observed. Death through *independent* acute intercurrent diseases is very rare; the victims of cholera Asiatica, typhoid (Peyerian) or typhus fevers, rheumatic fever, the exanthemata, acute Bright's disease, cerebral and abdominal inflammations, are scarcely ever the subjects of aortic aneurism.

(e.) Of coexisting chronic diseases, affections of the substance and valves of the heart, preceding, coincident with, or sequential to the arterial changes, are the most frequently conducive to the fatal issue: a rare example of death from phthisis now and again occurs; moderate emphysema is not very uncommon. Diathetic diseases of all varieties are uncommon; aneurism attacks people of the most vigorous frame and constitution.

833. *Repulsions and Affinities.*—In 108 cases of aneurism, Rokitansky found tubercle only five times—and always retrograde

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[1] Brader, U. C. H., loc. cit., into œsophagus. Whittaker, U. C. H., Females, vol. vi. p. 174, admitted immediately after violent hemorrhage, having the characters of hematemesis, rallied, had three more attacks within thirty-six hours, dying instantaneously in the last. The stomach contained fluid blood 7 oz., and two massy coagula weighing 13½ and 7 oz.; the sac, occupying the descending portion of the arch, communicated with the œsophagus, by an opening as large as half a crown.

tubercle. Twelve cases of aneurism of the arch, by Dr. Greene, supply four examples of tubercle; 132 cases of aneurism of the arch, collected by Dr. Crisp, furnish two examples of death by phthisis. Dr. Stokes teaches, on the other hand, that "the morbid condition which most often accompanies aneurism is tubercle. . . . I have often thought that there was a case deserving the name of consumptive or strumous aneurism, in which the same general morbid state, which caused deposition of tubercle in the lung, simultaneously affected the coats of the aorta." According, then, to Rokitansky, tubercle and aneurism are absolutely antagonistic; according to Dr. Stokes, of affinity so close that, in some cases at least, one and the same diathesis simultaneously generates both.

In sixteen cases of fatal aneurism of the arch, examined by myself *post mortem*, one rather doubtful example of tubercle occurs, in a male aged fifty-one; in one other male, aged thirty-two, there were pulmonary cavities, and growing tubercle; in all the other cases, the absence of crude tubercle or gray granulation is affirmed in my notes. On the other hand, I do not remember, in the vast number of phthisical people I have opened, to have met with any instance of progressive aneurism. Hence my contingent of experience supports the inference of Rokitansky as to the rare association of the two diseases. But the disparity of the ages most apt for the generation of tubercle and aneurism must not be forgotten; the mean age in my cases was forty-two; and it may be calculated that, omitting children altogether, about 63 per 100 of tuberculous adults, 13.5 only per 100 of aneurismal people, are under thirty years of age. The syphilitic cachexia and gouty diathesis are said to have some connection with aneurism: the fatty diathesis has possibly a stronger claim to the character.

A parallel, which I cannot help thinking somewhat fanciful, has been drawn between aneurism and cancer, in regard of their constitutional conditions and effects. Like the cancerous, it is said, the "aneurismal diathesis" is never extinguished: what comes, then, of the cures, spontaneous and by art, of the disease? Frequently, we are assured, many of the arteries are involved in the same person; the assurance is directly at variance with statistical returns: besides, admitting the fact, the analogy fails; cancers multiply through the blood; aneurism could only do so through local changes in the vessels. The aspect of the patient and the general decay of the organism resemble those observed in cancer, according to this argument: the statement is decidedly inapplicable to the majority of cases of aneurism, and only true of those attended with an unusual share of suffering. And, further, in their mode of distribution to the two sexes, aneurism and cancer are well nigh the antipodes of each other.

834. *Diagnosis*. Group *a*.—Locally considered, the diagnosis of sacculated aneurism of the arch turns essentially on the coexistence

of *a pulsating prominence*, visible and palpable, limited in area, and corresponding in seat to that portion of the vessel; the *signs of internal pressure; certain arterial murmurs and sounds;* and *limited percussion-dulness in the course of the arch.* But a pulsating prominence may also be produced by a small solid mass lying over and receiving the impulse of a sound aorta—the pulsation of a mass of the sort may even be quasi-expansile [807]; or it may be due to an abscess in the mediastinum; or to pulsating empyema; or to a tumor pulsating interstitially. Secondly, the signs of internal pressure may be produced by solid tumor. Thirdly, a double hoarse murmur over the arch, inaudible or only faintly audible at the heart—a diastolic murmur similarly localized, and a pumping or sucking character of the aortic sounds, without murmur, are the most significant auscultatory signs. Fourthly, several morbid states of the mediastina, bronchial glands, lungs, pleura, and even of the chest-wall, may accidentally render the percussion-sound dull in the course of the arch.

But the true difficulty in the diagnosis arises when not one of these physical signs exist with satisfactory distinctness; and when, in point of fact, the nature of the disease must be elicited on indirect evidence, and *per viam exclusionis.* The absence of symptoms and signs, indicative of ordinary affections of the heart and lungs, in an individual suffering from persistent anomalous disturbances within the chest, even though he does not, or rather because he does not, exhibit any failure of general health, affords strong motive for suspecting aneurism. If under such circumstances copious hæmoptysis occur, the diagnosis of aneurism, though undefended by a single positive physical sign, would rarely be at fault.

Tuberculous consolidation of one apex, especially the left, with murmur in the subclavian or pulmonary artery, is distinguished from aneurism by the non-extension of percussion-dulness across the middle line; by its extension, on the contrary, to the acromial angle; by the existence of some amount of tone in the percussion-sound, and some share of resilience in the wall of the chest; by the want of abrupt and sharply circumscribed definition of the dulness of percussion-sound; and by the absence of pressure-signs, eccentric or concentric. The symptoms, local and general, are also different.

Fluid in the pericardium is distinguished by the pyramidal form of its dulness, which aneurism never simulates except under the very rare accidental circumstances already referred to [479, *b*].

In a case of *enlarged heart*, there is but one centre of motion—in aneurism two; itself and the heart. When well-defined, this is a most valuable aid in diagnosis; but it must not be forgotten, that the aneurismal sac and the heart itself may be so closely contiguous, as to render it impossible to isolate them satisfactorily. Under these circumstances it may, or may not, be a matter of ease to detect such difference in the precise rhythm of pulsation at two given parts, as to settle the question in favor of a double source of motion. But we must remember that the sac may be not only non-expansile, but actually quiescent; under these circumstances the site of the percussion-dulness and the existence of pressure-signs must be appealed to for the distinction on the side of aneurism, and more or less of its special signs sought for on the side of enlarged heart. If general dropsy exist, this is in favor of heart disease, positively hostile to the existence of aneurism as the sole affection.

Pulsating empyema, with its throbbing prominence near the edge of the upper bone of the sternum, simulates aneurism closely, but may be distinguished by the rules which are laid down in the account elsewhere given of that rare form of disease.[1]

A chronic sub-periosteal abscess of the sternum, forming a small prominence in the line of the transverse portion of the arch, fell under my notice some time ago,[2] sufficiently resembling an aneurismal sac. But there was no impulse; gentle percussion immediately round the prominence gave normal resonance; there was no murmur, and concentric pressure-signs were totally absent. Still there might accidentally have been impulsive action of the vessel beneath, and excess of mediastinal fat might have rendered the percussion-sound dull; under such circumstances the diagnosis would have been excessively difficult.

Infiltrated cancer of the lung causes retraction of the side, produces no local prominence, deepens the intercostal spaces, and frequently renders the percussion-sound tubular in the infra-clavicular region; it does not produce pressure-signs, and may be the seat of the signs of softening and excavation.

Tumor in the anterior mediastinum presents the greatest number of positive points of similarity to aneurism. Now, if there be highly-marked pulsation, a broad-based prominence with conical elevation in the centre, the murmurs most distinctive of aneurism, and a sensation of the flow of liquid beneath the integuments, there can be no doubt that, whatever other grounds for diagnosing tumor may exist, aneurism is really present. But every one of these signs may in cases of aneurism be absent: then, observe how like the two things are—a sac filled with fibrin and a solid tumor. In truth, one is a tumor *inside*, the other *outside* the arch: the obstruction from without may have the same disturbing effect as from within on the blood-stream flowing in its interior. Common to the two things are dulness and non-resilience, usually extending across the middle line, all the signs of centripetal and all the signs of centrifugal intra-thoracic pressure. Under such circumstances, the question becomes one of pure probabilities. The conditions in favor of aneurism would be these: situation in the course of the arch; vibratile thrill above or below the clavicle; gradually increasing nearness of pulsation to the surface;[1] double impulse, especially with doubling of diastolic share of impulse; dysphagia; great pain, especially of the dorsal spine; absence of œdema of the arm and chest. The circumstances in favor of tumor, and against aneurism, would be the facts of the patient being a female,[2] and under twenty-five years of age; great superficial extent of percussion-dulness, especially if there were no marked attenuation of the walls of the chest; absence of any heaving motion in the affected spot; want of accordance between the sites of maximum dulness and of pulsation; and currant-jelly expectoration common with tumor, very rare with aneurism.

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[1] Diseases of the Lungs, 3d Am. edit., p. 241.

[2] Marg. Motlee, U. C. H., Females, vol. v. p. 36.

It is a curious fact, that where a *quiescent aneurismal sac* and a *tumor* coexist, the usually essential sign of aneurism, namely pulsation, apparently expansile, may be furnished by the tumor; and the usually essential sign of tumor, dead, pulseless dulness under percussion, may be caused by the aneurism. This statement is well illustrated by the case of Brader (*loc. cit.*).

The means of distinguishing *coarctation of the arch* will be found with the description of that disease.

Aneurism of the innominate artery is distinguished by the higher position of its pulsating prominence behind or above the inner part of the clavicle; prominence appears relatively early; dysphagia, tracheal pressure-symptoms and dyspnœa are comparatively rare; the clavicle is often pushed from its place; paralytic symptoms in the right arm are greatly more frequent; the respiration-sounds are seldom enfeebled—if they are so, the right lung suffers. In an elaborate essay by Dr. Holland,[3] I find it stated, that the arteries in the right side of the neck and head and in the right arm generally pulsate less strongly than on the other side—whereas the reverse is the general fact in cases of aneurism of the transverse portion of the arch.[4] The same writer notes that pressure on the right subclavian and carotid diminishes or stops the pulsations of an innominate sac—while, brought to bear on either the right or left arteries, it exercises no influence on aortic aneurismal action.

835. Group *b.*—*Fusiform dilating aneurisms.* The peculiarities of these aneurisms are, as compared with the sacculated, diffuseness of pulsation above and below the clavicle, visible and palpable—comparatively much less below than above, though even there, if anæmia exist, it may be very considerable; more thrill above, less below, those bones;[5] rough, prolonged, rasping, whizzing or whirring

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[1] Yet, from stratification of fibrin, the pulsation of an aneurism may grow deeper; and, on the other hand, that of a tumor may become more superficial.

[2] But this is of little value, for the excess of aneurism of the arch in males is by no means so great as that of aneurism of all arteries indiscriminately.

[3] Dublin Journal, 1852.

[4] This rule is, however, open to numerous exceptions; in my own cases of ascending and transverse aortic aneurism combined, the weakly acting radial was more frequently the right.

[5] But thrill may be totally absent in this form of dilatation, as proved *post mortem; e. g.*, Fullaway, U. C. H., Males, vol. xvii. 1860.

murmur, systolic only, audible along the arch, and louder there than at the aortic valves, if they also be the seat of murmur. Centripetal and centrifugal pressure-signs are almost or completely wanting.

Anæmia and nervous excitement, by causing violent throbbing action, may simulate such dilatation; but the results of percussion will distinguish the cases.

836. Examples of *globular dilatation of the arch* are so rare, that a note of the principal phenomena, observed in the only case I have seen, will not be misplaced:—

Mr. ——, æt. 50, January 13, 1852; intense pain, upper sternal and left infra-clavicular region, radiating to axilla and elsewhere variously; no murmur over heart, nor aorta in front; distinct systolic short superficial weak murmur in course of thoracic aorta, just at the level of the inferior angle of the scapula; no prominence or impulse visible in infra-clavicular region; no thrill above or below the clavicles; percussion dull in left inter-scapular region, and slightly so even in the right, also about first and second left costal cartilages in front, but patient so sensitive that careful percussion impossible. Respiration in left inter-scapular region, and below this, peculiarly jerking,[1] of very sharp blowing quality. March 31; impulse now visible, as also very slight general prominence about the top of the sternum, and the two upper left costal cartilages; no thrill; no murmur in heart or aneurism anteriorly; double sound over prominence, both divisions louder than at the heart. April 26; prominence more obvious, and area of dulness increased; absence of thrill or murmur as previously.

This patient destroyed himself a few days after the last interview, all the measures employed failing to relieve his pain. The aneurismal dilatation was of large size (U. C. Mus., No. 4036).

837. Group *c.*—*Mixed aneurism.* The sudden extension of dulness in the situation of a simple sac, coupled with similar increase of pressure-signs, especially if these conditions follow effort of any kind, makes it probable that the inner and middle coats have given way, and the outer undergone additional pouching. But in the signs of a mixed aneurism, when actually developed, there is nothing special. The prognosis is rendered worse by the yielding of the inner coats.

II.—ANEURISM OF THE DESCENDING AORTA.

838. The signs of an aneurism seated between the termination of the arch of the aorta and the diaphragm will, of course, vary somewhat with the precise portion of the vessel affected. If the sac be not of large dimensions, little is to be learned by inspection; however, in certain positions, in consequence of its lying behind the heart and pushing this organ directly forwards against the ribs or sidewards, the maximum cardiac impulse may be transferred from the apex to the base, and so-called diastolic impulse also produced. Posteriorly the hand may detect slight arching to the left of the

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[1] The sac, being exceedingly loose, probably caused the jerking rhythm of the respiration by pulsating against the main bronchus.

spine; if the arched surface be the seat of the least impulsive action, the sign becomes one of importance; but arching may be totally deficient, even though the sac be large.[1] Dulness under percussion, limited to the same situation, and inexplicable by the condition of the lung, heart or pleura, would, of course, strengthen the inference drawn from the previous sources. An aneurism in this situation may supply the varieties of murmur and sound already enumerated; murmurs must be stronger over the sac than over the heart, to have any diagnostic value. Feebleness or deficiency of respiration close to the spine, or over the side generally from pressure upon the main bronchus, will corroborate the evidence of the other signs.

A case, observed some while since, revealed to me the unexpected fact that every one of the physical signs of aneurism of the descending thoracic aorta may exist to the *right*, instead of to the *left*, of the spinal column posteriorly. In this singular case, marked pulsation, percussion-dulness, and a number of other signs as well as symptoms (free hemorrhage among the number), pointed to the existence of a large aneurismal sac. Yet, so complete was the nullity of physical signs to the left of the spine, that, although well aware of the possibility of vessels becoming twisted out of their normal line and site by the growth of an aneurismal sac,[2] I could not bring myself to diagnose that affection with positiveness, vacillating between it and a carcinomatous tumor receiving the impulse of the vessel. On *post-mortem* examination a sac of considerable size was found, lying to the right of the spine, and having actually dragged the trunk of the vessel across the middle line in the process of its own enlargement.[3]

839. Vertebral gnawing pain and intercostal neuralgia are occasionally observed. There is sometimes a subjective sense of throbbing action, which is with difficulty distinguished from that of the heart.

The laryngeal system commonly escapes. This habitual freedom obviously depends on non-implication of the recurrent nerve, and on the mere distance of the larynx from the locality of the disease. Sometimes, however, the larynx suffers through extension of irritation from a main bronchus; the trachea, indeed, has, in rare instances, undergone direct pressure from very large-sized sacs. Perforation of the main bronchus, dysphagia from mechanical obstruction of the œsophagus, or, where the disease occupies the immediate vicinity of the cardia, various gastric symptoms simulating internal obstruction of that orifice of the stomach, have been noticed.

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[1] Moriarty, U. C. H., Males, loc. cit.

[2] This is far from being very uncommon in aneurisms of the arch of the aorta in front.

[3] Case seen once, within a few days of the fatal termination by hemorrhage, with Dr. Allchin. The thoracic aorta, but without the spine, is now in University College Museum, No. 4602.

840. Death occurs more frequently, perhaps, from rupture into the œsophagus than from any other single cause; curiously enough, rupture into the right occurs almost, if not quite, as frequently as into the left pleura. The patient dies, worn out by the effects of bronchial and tracheal pressure in some rare cases.

841. Of special causes for the disease in this particular situation nothing is known. It is noteworthy that aneurism of this division of the aorta is singularly rare in females.

842. The affection with which an aneurism, thus seated, may most readily be confounded, is hypertrophy of the heart; the site of the strong systolic and diastolic impulse may readily deceive. But careful employment of all the methods of physical diagnosis will prevent error in well-marked cases; while it must be confessed that very small sacs behind the heart, unless some accidental circumstances throw light on their existence, are exceedingly difficult of positive detection. The obscurity occasionally arising from displacement of the sac and connected vessel to the right, must not be forgotten.

843. *Duration and prognosis of aneurism of the arch and descending thoracic aorta.*—There is an unfortunate deficiency of materials on any large scale for determining the mean clinical duration of the disease. That, once developed, it will eventually destroy life is an inference practically unshaken by the few recorded examples of alleged cure. But it is astonishing how long life may be prolonged, even with tolerable comfort to the individual, by such simple management, hygienic and medicinal, as common sense dictates. I shall not easily forget the case of a man first seen in November, 1846, with a large aneurismal prominence, pulsating so liquidly, if I may use the word, and so directly under the skin, that it became necessary to apply a mechanical protection against the danger of rupture from a blow or even from sharp friction—a man worn with suffering—unable to sit, lie, or stand, from pain and general uneasiness—and wasted considerably in flesh and strength: and yet the death of this patient did not take place till July, 1849—he having, meanwhile, though actively phthisical also, lost the major part of his more serious sufferings, and, in a certain subdued fashion, actually enjoyed existence.[1]

It is positive that the mean duration is greater when the sac grows centrifugally than centripetally.

844. *Treatment.*—In proceeding to the treatment of an aneurism of the thoracic aorta, the first point is to determine, if possible, whether we have a fusiform or globular dilating aneurism, or one of the sacculated varieties, to deal with. For, whilst coagulation of the blood within the aneurism is scarcely attainable in the former cases, and if obtained will not effect their cure; in the latter, there is a natural tendency to such coagulation, and coagulation does

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[1] Harris, U. C. H., Males, vol. ii. and viii.

actually promote anatomical cure. Hence it appears that the clinical distinction of these kinds of aneurism is not a piece of mere scholastic refinement, as it has been slightingly called, but a matter of practical importance.

There is another point worthy of serious consideration. With what hope of achieving a cure do we undertake the treatment of the case? If, as experience amply shows, existing methods have seldom, if ever, accomplished removal of the disease, clinically understood, risk, even the slightest, should obviously be avoided of doing mischief to the constitution by the energetic employment of any one of those methods.

(*a.*) In cases of fusiform and globular dilatation, the indications are the prevention of enlargement and rupture of the aneurism. These ends may be best secured by occasional leeching over the affected part, especially if there be local tenderness—much better than by small bleedings from the arm. Full and repeated venesection, on the plan of Valsalva, is in this species of aneurism even less permissible than in the sacculated varieties: but a single abstraction of some eight or ten ounces of blood at the commencement of treatment, more especially in plethoric persons, sometimes gives considerable relief to distressing symptoms within the thorax—even this should be cautiously done, however, if the system at large has at all suffered from the disease. Purgatives, to such amount as to maintain a tolerably constant free action from the bowels, both from their sedative effect on the circulation, and from their preventing the necessity for effort in defecation, are essential. Diuretics do service by preventing the water in the blood from rising above par. Direct sedatives of cardio-vascular action, digitalis, aconite, hydrocyanic acid, and belladonna, internally and externally, lessen the violent pulsatile action of the diseased vessel, and may be given in various combinations, and more or less steadily.

The diet should be so arranged as to support, without exciting or over-nourishing: but anæmia is even more baneful than plethora, and the starvation system must be studiously avoided. The patient should abstain from all excitement, mental and emotional, pass the greater part of his time in perfect rest, and take but moderate daily exercise on foot: carriage exercise on smooth ground may be permitted to any amount desired.

(*b.*) In the instance of a sacculated aneurism, the object being to promote coagulation, occasional venesection, with the view of lessening the force of the current, is commonly recommended. But there is a double danger to avoid here: if too much blood be drawn, the action of the circulating system will be excited, instead of tranquillized; and if the quality of the blood be seriously impoverished, the softness of the coagulum will probably render it comparatively useless as a support to the distended walls of the vessel. Theory, in truth, does not, at the present day, support the ideas of Valsalva concerning abundant depletion; and since the diagnosis of aneurism has become somewhat positive, cures obtained by his system have ceased to be heard of. True, the recommendations of Valsalva are rarely, if ever, executed to the letter: the courage of patient and physician generally fails—in time, probably, to prevent irremediable mischief. Moderate leeching over the sac from time to time is generally useful. Digitalis and other cardiac sedatives promote coagulation by enfeebling and slackening the current; and if there be no contra-indication in the state of the heart, a fair trial of them should never be omitted. Some practitioners have much confidence in the acetate of lead. Purgatives and diuretics are useful on the principles a moment since referred to; the latter especially, because, while they diminish the water, they exercise no influence on the fibrin, of the blood.

Cold poultices of linseed meal and vinegar, or of conium and digitalis, relieve local suffering, and probably promote coagulation.[1] Ice to the surface is grateful to some patients, unbearable by others: it can rarely be kept applied for a sufficient length of time to modify the circulation beneath. Cold poultices of oak bark have appeared to me useful. If there be excessive pain over the sac, the application of the freezing mixture, as recommended for anæsthetic purposes generally by Dr. James Arnott, would, for a double reason, deserve a cautious trial. The local application of chloroform I have also found useful. Counter-irritation in the neighborhood of, but not over, the sac sometimes relieves greatly. I have known interscapular pain completely disappear under the use of caustic iodine.

Tannic and gallic acids, combined with digitalis, aconite, or belladonna, have appeared to me to exercise a very beneficial effect in promoting coagulation: gallic acid may be given in doses of three, five, and eight grains twice or thrice daily, with occasional intermissions, for a length of time—its constipating effects (if such really arise) being obviated by occasional doses of castor oil.

The theoretical necessity for fibrin of good quality being clear in these cases, such diet is advisable as seems to promote its formation. However, the dangers of plethora, especially as much exercise cannot be permitted,[2] must be held constantly in view, and over-nourishment avoided. Fluid in any quantity is injurious, and stimulants seriously baneful.

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[1] But the temperature must not be too low; coagulation is prevented by cold = 40° Fahr.

[2] No argument in favor of exercise appears to me to flow from those exceptional cases, in which violent physical exertion is borne, at the moment, with impunity. Thus it is well known that the celebrated and lamented surgeon, a few years since carried off from among us by aneurism of the transverse portion of the arch, some time after he had lost quarts of blood from the sac, and while already seriously distressed by tracheal pressure-symptoms, performed feats of personal prowess, pedestrian and other, not only without immediate ill results, but, as he himself maintained, with relief to his sufferings. The ultimate and real effect of such strain upon the circulation must have been to hasten the enlargement of the sac.

Galvano-puncture, as originally suggested by Pravaz for external aneurisms, has occasionally been thoroughly successful, even with vessels of considerable calibre—as the external iliac:[1] but occasionally the results have been disastrous—and the application of the plan to the aorta would be so imminently hazardous in various ways as to be unjustifiable. The same may be said of the injection of a few drops of the perchloride of iron; but the free administration of the salt internally, as prepared by Burin for M. Pravaz, seems worthy trial.

If the aneurismal sac be very superficial, and its wall ill-protected by fibrin near the parietes of the chest, it may be necessary to apply a shield, fitted to the part, to protect it from the chances of external violence.

The laryngeal symptoms of aortic aneurism have frequently been mistaken for evidences of disease in the windpipe itself, and tracheotomy vainly performed for their relief. Dr. Gairdner, however, endeavors to show that the operation presents a fair claim to be admitted into the legitimate treatment of aortic aneurism—"not unwillingly and as a last resource, but as early as it could be ascertained that laryngeal symptoms were the source of the more immediate danger." Dr. W. Begbie, in turn, advocates the proceeding, on the ground of its converting a very painful into a comparatively quiet death.[2] I have had no personal experience of the operation in cases of aortic aneurism; but can conceive it might become justifiable as a means of *probably* prolonging life for a few hours, where such prolongation might be a matter of importance.[3] And if the symptoms are purely those of spasmodic laryngismus, considerable relief might be temporarily obtained: under these circumstances, the operation was indeed many years ago recommended by Marshall Hall.[4]

Arguing from the "extraordinary relief" to symptoms often afforded, when an enlarging sac, compressed by the clavicle, at length succeeds in partially dislocating the bone forwards, Dr. Stokes suggests that, where such displacement of the bone failed to take place spontaneously, division of the sterno-clavicular ligaments might be advantageously effected with the knife. I have certainly seen instances in which spontaneous luxation seemed to lessen the amount of suffering of these patients. In one instance, in particular, where the displacement of the sternal end of the bone was sufficient to put the attached portion of the sterno-mastoid muscle "on the stretch," the patient felt "getting better and better every day," and as though "nothing were the matter with him."[5]

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[1] Eyre, in Lancet, July, 1853.

[2] Ed. Med. Journal, 1858.

[3] A case recorded by Mr. Judd seems to show that life may by possibility be prolonged for several days, nearly a fortnight, by tracheotomy—though the trachea suffers very serious pressure from a large sac; but obviously, we have no right to expect the repetition of so extraordinary an occurrence (Lancet, 1844).

[4] Lancet, Nov. 1852.

[5] Covey, U. C. H., Males, vol. xi. p. 316.

In not a few cases of aortic aneurism the treatment becomes less that of the disease itself than of some associated state, as hypertrophy, or flabby weakness of the heart, or anæmia—or of some sequential affection, such as obstinate bronchitis. Tartarized antimony, nitre, and digitalis in combination, are the best remedies for the latter malady.

III.—ANEURISM OF THE ABDOMINAL AORTA.

845. *Physical signs.*—Inspection may, or may not, disclose some abnormal appearance. If the sac be small, and especially if it spring from the posterior aspect of the vessel, or if the aneurism be of the fusiform species and not bulky in any part of its extent, the eye may fail to detect any peculiarity in the form of the abdomen. If the sac have acquired any size, pulsating prominence, of variable extent, is seen anteriorly, in the course mainly of the aorta, from the epigastrium downwards,[1] or bearing especially to the left side of the abdomen, in rare instances to the right. The surface is smooth to the eye; the respiration-movements laterally, or bilaterally, impeded.

The hand, placed on the anterior prominence, receives a single systolic impulse, sometimes of enormous force and quite out of proportion with the volume of the moving mass, while posteriorly, in the lumbar region, no trace of impulse may be perceptible. Generally speaking, if the abdominal walls be thin, the hands may be passed on either side of the sac, and an estimate formed of its bulk: the impulse is felt to be laterally, as well as anteriorly, expansile. The chief pulsation may be to the right of the spine, the sac sometimes mainly growing in that direction. The mass, fusiform, rounded, smooth or lobulated, is immovable,[2] and commonly compressible more or less: caution is, however, requisite in ascertaining these particulars. In comparatively rare instances a second impulsive, or at least strongly jogging, action, diastolic in time, may be felt: or, on the other hand, pulsation may be absent when the sac is quite large enough to alter the form of the abdomen slightly; while, *per contra*, a very small sac sometimes furnishes powerful impulse. Thrill, systolic in the majority of cases, of diastolic time in rare instances, may be felt.

The left semi-circumference of the abdomen may or may not be increased.

The size of the sac can only, if at all, be accurately estimated by percussion; and the tenderness of the surface and the neighboring parts generally often interferes with the process. Percussion of any force is dangerous and unjustifiable—especially as intestinal note habitually interferes with precise limitation, both vertically and horizontally [99]. Besides, a considerable time before death, the apparent size of the sac may be greatly increased by successive and repeated extravasations of blood behind the peritoneum.[1]

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[1] Hallington, U. C. H., Males, vol. viii. p. 61. The visible prominence here extended from one inch to the right of the umbilicus, to the anterior spine of the left ileum: dilatation of the aorta commencing an inch below the origin of the renal arteries; sacculation implicating the lower part of the aorta and the common iliac.

[2] In rare instances the mass is movable; and in such cases, Dr. Stokes points out, pulsation and even murmur may be made to appear and disappear.

I have heard in connection with aneurism thus seated: 1. A single systolic murmur, without sound of any other kind. 2. A dull muffled systolic sound, convertible into a murmur by a little pressure. 3. A sharp, abrupt, short systolic murmur at the left lumbar spine, much more marked than in front. 4. A systolic murmur below the sac, none immediately over it. 5. Occasionally a dull second sound. I have never heard a murmur diastolic in time. In some instances systolic murmur is audible in the reclining, when inaudible in the erect, posture: Dr. Corrigan supposes that in the earlier periods of the disease the hydrostatic pressure from above, by maintaining a state of tension of the sac, prevents the occurrence of murmur; while the diminution of pressure in decumbency, lessening the tension of the sac, allows of its production. Sometimes a moment's decumbency suffices to bring out the murmur well; at others the lapse of two or three minutes is required.[2] But I agree with Dr. Stokes, that this peculiarity will not help to establish the diagnosis between pre-aortic tumor and aneurism.[3] Besides, it is indubitable, true aneurismal murmur sometimes disappears in the sitting and standing postures. The student must further remember that murmur may be totally absent in every possible posture.

The special characters of murmur vary as in the arch of the vessel: its amount sometimes changes inversely as the growth of the aneurism. It may be so loud as to be audible at a little distance from the surface.[4]

It is all-important for the observer of an obscure abdominal disease to bear in mind that even large-sized aneurism of the ventral aorta may exist in the total absence of all positive physical signs—neither impulse, murmur, nor percussion-dulness being discoverable: the subjective symptoms are then very likely to deceive.

846. *Symptoms.*—The patient may or may not be conscious of pulsation, and this whether there be or be not, objective evidence of morbid action of the kind. Subjective pulsation, absent for two years, in a case observed by myself, then suddenly came on after

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[1] The extravasated blood may make its way to the front of the abdomen, and extend upwards to the pleura. Hallington, U. C. H., loc. cit. The extravasated coagula may pulsate distinctly under the influence of the sac, and with expansile character. I leave this passage exactly as it stood in the last edition. Dr. Stokes (op. cit., p. 627), in observing that I state "the secondary tumors do not pulsate," has by some accident misinterpreted my words.

[2] Dub. Journal Med. Science, vol. ii.

[3] Dis. of the Heart, p. 643. Vide Appendix.

[4] Case by Dr. Reynolds, "Med. Times," Sept. 1852, p. 284.

effort in running, and continued until death. Pain following the course of nerves implicated by pressure—passing along the edge of the ileum down the thigh to the testicles and pudenda generally—and in character raw, sore, pricking, cord-like, plunging, hot and burning at one time, cold at another, accompanied with spasmodic difficulty in passing urine, and with tonic contraction of the flexor muscles and inability to straighten the limb, the whole attended also with peculiar gnawing vertebral pain, existed in the case already more than once referred to. But obviously the neuralgic sufferings must vary with the exact site of the sac; sometimes they are relieved by special changes of posture. Theoretically, anasarca of the lower limbs, or of one of them, must occur, according as the inferior cava or either iliac vein is pressed on; but in practice either effect is most rare—and fulness of the subcutaneous abdominal veins from obstruction of the cava may exist without any pedal œdema even. Wasting of the testicle I have seen from obliteration of the spermatic artery. Pressure on the descending or transverse colon may obstruct the bowels, and cause flatulence, constipation and great labor in defecation. In the case of Hallington (loc. cit., p. 64) the sac was adherent to the descending colon by pseudo-cellular bands, and the calibre of the bowel in one spot much reduced by contraction of these bands. Ascites of any clinically important amount is excessively rare, if indeed it ever occur through the influence of aneurism alone. The rarity of dropsy of any kind is in regard of diagnosis seriously significant.

The respiration, if the sac be of moderate size and low down, is of natural frequency and character; when high or of notable bulk it interferes with phrenic action, throws the onus on the upper ribs, and accelerates the act somewhat. Cough may be excited by mere pressure from below; but hæmoptysis does not occur, unless there be perforative communication with the lung.

The pulse, even while the patient is visibly perishing from pain, insomnia and exhaustion, need not be accelerated—ranging from 78 to 88: the pulse-respiration ratio may be a normal one of 4.35 : 1. The heart may be raised upwards or pushed sidewards.

The urine may be rendered albuminous by renal congestion induced by pressure on the emulgent vein; otherwise it is perfectly natural, as far as the aneurism is directly concerned; but when the constitution begins to sympathize, I have observed continuous oxaluria.

Professor Seaton Reid has published a curious case, in which an aneurism arising opposite the cœliac axis and separating the pleura from the diaphragm, seemed to be the only apparent cause (through traction of the great splanchnic nerve on the right side of the sac), explanatory of contraction of the right pupil.

847. *Duration, and mode of death.*—In some instances these sacs have acquired enormous bulk: one preserved in the Fort Pitt Museum is said to have contained ten pounds' weight of coagula.

Hence the inference that the gradual growth of the disease is not incompatible with existence. But we have little information as to the mean duration of life after the outbreak of symptoms: I have known twenty-four, thirty, and thirty-eight months intervene between the earliest indications and the fatal termination.

Death occurs by rupture of the sac behind or into the peritoneum, into the pleura, lung, colon, renal pelvis, or mediastinum; or without rupture by jaundice, gangrene, exhaustion, &c.

848. *Prognosis.*—People with aneurism of the abdominal aorta have in rare instances escaped that fatal termination, which is almost the necessary apanage of the disease. The sac has filled with stratified fibrin, and symptoms have wholly, or almost wholly, disappeared. Such patients have not then actually died of their aneurism, though unquestionably its existence may have rendered them more ready victims, than they otherwise would have proved, of the disease actually destroying them. Complete repletion of a sac with coagulum, even to the level of the normal interior of the vessel, does not insure removal of symptoms. All the evils, obstructive of function and excitant of pain, depending on abdominal tumor, may be felt as vividly as before; this has more than once been proved by *post-mortem* examination.

849. *Diagnosis.*—The diagnosis may be conveniently considered under the three heads of cases: (*a*) where there are abdominal physical signs; (*b*) where there are no physical signs, but serious ventral symptoms, mainly neurotic, with or without much constitutional sympathy; (*c*) where there are no signs, or very trifling ones, and no symptoms of the least apparent consequence.

(*a*.) The extreme difficulties sometimes arising in the distinction of mere *aortic pulsation* from aneurism have already been considered [807]. Curiously enough, the only case in which I have heard abdominal aortic murmur, diastolic in time, was one of diminished, instead of increased, calibre of the vessel. *Fecal accumulation* is distinguished generally by the oval outline of the fulness; by its doughy inelastic feel; by the existence of several spots of dull and clear resonance under percussion close to each other, and within the area of the swelling, from the intermixture of gas with solidified feces; sometimes from the position of the mass; and, generally, from the history of the case. The pains of aneurism may be imperfectly imitated by those of peritoneal distension from the enlargement of the bowel; but it is rare indeed that a mass of feces receives such arterial impulse from behind as to simulate that of aneurism.—In the obscurity of their early symptoms, in the eventual pain, and in the gradual exhaustion they produce, there is considerable similarity between *lumbar and psoas abscesses* and aneurism; but the swelling of these abscesses passes in an elongated form from above downwards, and does not exhibit an irregularly globular shape, as aneurism sometimes does; they give neither impulse nor murmur. Tenderness exists in the lumbar spine, and there may be loss of motor power in the lower extremities; but the actual pain is materially less, as a rule, than in the aortic disease. Tubercles should be sought for in the lungs: their presence would be directly in favor of lumbar abscess of tubercular origin —against aneurism.—*Hydronephrosis and pyelitic distension* are accompanied with renal symptoms, changes in the urine, tumor with the characters of renal enlargement—a tumor of tuberous nodular outline, non-impulsive, murmurless, and extending further into the flank and into the back than aneurism. The urine may be albuminous in all three affections.—*Tumors* of various kinds in the abdomen may pulsate in expansile manner, and be the seat of murmur; the murmur is high-pitched, whiffing invariably as far as I have heard; but a careful consideration of the whole case is the best safeguard against error. In a case of cancerous lumbar glands, seen some time since, not only were there expansile impulse and murmur, but such neuralgic pain as commonly accompanies aneurism; still the knowledge that a cancerous sarcocele had previously been removed prevented mistake. When there is an obvious solid mass, either a tumor or an aneurism, and which is pulsatile in the recumbent posture, the diagnosis may sometimes be made by placing the patient on the hands and knees; if aneurismal, the pulsation will remain; if that of tumor, conveyed from a healthy aorta, it will disappear, in consequence of the vessel and the morbid mass having ceased to be in close juxtaposition. But the presence of adhesion would greatly interfere with the employment of this test.—*An enlarged lumbar vertebra*, pressing forward the aorta, will cause extra pulsation; but there is no lateral expansion of the vessel; and the murmur is whiffing or rasping, and not heard laterally.[1]

And even if there be positive surety of the existence of abdominal aneurism, we are not at once entitled to pronounce it aortic; it may be situated in the cœliac axis, the hepatic, the superior mesenteric, or the renal arteries. But the signs of these aneurisms have as yet been imperfectly investigated,[2] and I know nothing of any one of them by experience.

(*b*.) Wherever obstinate abdominal neuralgic pains exist, especially in a male, and where the ordinary signs of visceral disease cannot be established, aneurism should be held in view as most probably present, even though there be no single physical sign to warrant such an opinion. Let the examination never be considered complete, however, without careful auscultation in the left vertebral groove. It will be necessary, too, to exclude, with as much certainty as possible, the presence of cancerous lumbar glands. An aneurismal patient of this class may be seen at a time when his general health is excellent, or already impaired.

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[1] Vide case of Dr. Taylor, in Author's work on Cancer, p. 528.

[2] Vide Ballard, Physical Diagnosis of Dis. of the Abdomen, p. 217.

(*c*.) Slight pain in the lumbar region may be the sole symptom for twelve months, to my own knowledge, after aneurismal distension has probably commenced. Hence the importance of thoroughly investigating physically cases of alleged incurable lumbago and sciatica. Relief of lumbar pain by cupping will not disprove the dependence of that pain on aneurism.

850. The treatment is the same as of intra-thoracic aneurisms in general. Were the disease diagnosticated at an early period, might any good be effected by pressure either on, above, or below the sac?—a cautious trial of one or other form of pressure might with propriety be made.

Rest, as complete as can be managed without serious disturbance of the digestive organs, is, beyond doubt, of extreme importance in cases of the class. There are many instances on record in periodical journals, where perfect quiet so singularly improved the local and general state as to make the patient believe himself free from disease, and consequently to commit acts of imprudence which brought back the previous symptoms with increased intensity.

851. Group *d. Dissecting Aneurisms.*—The morbid anatomy of dissecting aneurism of the aorta, in its three essential varieties, is clearly demonstrable from existing records: its clinical history has yet to be worked out. And, indeed, from the nature of things it seems singularly unlikely that any general account, applicable even to the majority of such cases, can be given—seeing that the symptoms must in great part depend upon the extent and precise portion of the aorta affected.

852. The symptoms in published cases may clearly be referred to three heads, which the observer should always aim at severally distinguishing;—namely, (1) symptoms of shock to the system at large; (2) of dynamic and statical disturbance of the injured artery; and (3) of mechanical interference with the function of organs supplied by branches from the injured part of the vessel. (1.) The symptoms of shock are, primarily, sudden faintness or actual syncope, and, on recovery of consciousness, nausea, vomiting, and pain in the thorax or abdomen: secondarily, febrile action, by no means necessarily very marked, thirst, furred tongue, abdominal tympanitis. (2.) The dynamic disturbances of the artery are signified by more or less severe pain in its course, and throbbing action, irregular in force and rhythm. Statically, the vessel and its injured branches are widened and rendered uneven; while the obstruction to the current, offered by the prominent and ragged lining membrane in the site of its ruptures, gives rise to blowing systolic murmur, which, if seated near the heart, may be mistaken for that of constrictive disease of the aortic orifice. (3.) The symptoms of mechanical origin are produced by the accumulation of the blood, filtrated between the coats of the aorta, against the orifices of arterial branches, whereby these are completely, or almost completely,

blocked up. The nature of these symptoms will, of course, depend on the distribution of the blocked-up vessels. Thus, in a remarkable case, observed by Dr. Todd (*Med.-Chir. Trans.*, vol. xxvii.), where the innominate and the renal arteries were mainly obstructed, very singular cerebral symptoms and suppression of urine marked the event. If a main bronchus were pressed on by the suddenly enlarged vessel, equally sudden deficiency of breathing (the percussion-sound remaining unaltered), would ensue.[1]

In not a few of recorded cases there were no conditions of the heart or great vessels known to exist prior to the actual event, which might have led the observer even to suspect such dissection of the arterial coats as a likely or even intelligible occurrence. In certain rarer cases the existence of such previous diseases has been known, and would certainly aid in the diagnosis. This is especially true of certain forms, as, for instance, where the arch of the aorta was known to be the subject of serious coarctation [864].

853. Were the practitioner fortunate enough (guided by the sudden supervention of symptoms of the three classes just distinguished, and of a strong arterial murmur in a person known to have previously been free from this physical sign) to divine the occurrence of acute separation of the coats of the aorta, it does not appear that, in the present state of knowledge, the treatment would be materially improved by his sagacity. Did he fail to diagnose the occurrence, his aim would be to recover the patient from the first shock of the accident, control excited arterial action, and relieve symptoms as they arose. And it does not appear that art could do more than this, were the anatomical nature of the affection understood from the first.

IV.—VARICOSE ANEURISMS OF THE ARCH OF THE AORTA.

A.—COMMUNICATION WITH THE SUPERIOR VENA CAVA.

854. A woman, aged fifty, having suffered for some years from aneurismal symptoms, one evening while stooping at laborious work, suddenly felt as if strangled, changed color, felt giddy, and sat up all night in dread of suffocation. When seen the next day, the face and upper part of the body were deeply cyanosed, the tributaries of the upper cava enlarged, those of the lower natural. The ordinary signs of aneurism of the arch existed about the right infra-clavicular region. Dr. Mayne, the observer of the case, arguing from the existence of a powerful superficial whirring systolic murmur, loudest at the second right cartilage, accompanied with thrill, perceptible not only at the spot, but, in spite of great œdema and swelling, over the right internal jugular and subclavian veins, and taking into consideration the extreme suddenness with which the serious symptoms above referred to set in, made the diagnosis

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[1] Vide also a case of Dr. Risdon Bennett, Med.-Chir. Trans., vol. xxxii.

of an aortic aneurism communicating with the superior cava. The pulse full, strong, and jerking, beat about 110 in the minute.

Death ensuing on the tenth day from the patient's sudden seizure, an opening "in size and shape resembling the button hole of a shirt, and crossed about the centre by a delicate frenum," was found between the vena cava and an enormous globular dilatation of the arch.[1] The heart exhibited nothing abnormal: the jerking pulse, it may therefore be presumed, depended directly on the loss of arterial current, sustained through escape of blood from the sac into the vein; but such mechanism is so singularly rare that the occurrence of jerking and visible pulse in connection with it can scarcely be held to invalidate the significance of the sign in relation to aortic reflux.

B.—COMMUNICATION WITH THE PULMONARY ARTERY.

855. If an individual, known or not known to have been the subject of aortic aneurism, suddenly experience after effort a sensation of something giving way in the cardiac region, feel faint, become pale and exhibit the general characters of nervous shock to the heart, followed by peculiar fluttering in the chest—if he subsequently suffer from dyspnœa to orthopnœa, more or less cyanotic blueness of the lips, pallor of the face, chilliness, prostration of strength, anxiety, terrible dreams, occasional nausea and vomiting, syncopal and pseudo-epileptic attacks, and become anasarcous in the lower extremities, while the lungs and liver undergo mechanical engorgement, as proved by percussion—if all this coexist with powerful systolic thrill, limited to the second and third left interspaces close to the sternum,[2] and with loud whirring murmur essentially systolic and intermittent, though sometimes inclining to continuousness (or, it may be, double murmur similarly seated), the diagnosis of sudden communication between the aorta and either the pulmonary artery or right ventricle is warranted. The further determination of the existence or not of aortic aneurismal dilatation must turn on its own special signs.

Dr. Wade has published a case from which he appears to infer that non-conduction of existent diastolic murmur downwards to the heart's apex is the key to the diagnosis of aortic communication with the pulmonary artery. But this inference seems to me very seriously shaken by the fact that in some cases of aortic regurgitation the attendant diastolic murmur is not transmitted to the left apex—where, on the contrary, a pure second sound is heard: this holds true even of excessively loud basic reflux murmur [149, 718].[3]

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[1] Dublin Hospital Gazette, Feb. 1854.

[2] This limited site will be lower in position, somewhat, if the heart itself be lowered by hypertrophy.

[3] Proceedings Med.-Chir. Soc. 1861.

Recorded cases[1] are, as yet, too few in number to supply trustworthy information concerning the possible duration of life after the communication has been accomplished. A history, printed by Dr. Smith, seems to show that existence may be prolonged for three months; here the patient died in a pseudo-epileptic fit.[2] I have before me a preparation in which the vessels communicate by an opening, round, smooth, and half an inch in diameter on the aortic side; round, of the size of a split pea on the pulmonary side:[3] according to the catalogue, the existence of the aperture was "not detected during life by any morbid sound or symptom." And it is remarkable enough that in Dr. Wade's case the symptoms, at the moment the communication between the two vessels was probably effected, were but slight: and the patient left the hospital after "a two or three weeks'" sojourn, "declaring himself well." He eventually perished about a week afterwards from a fresh rent, extending from the sac into the right ventricle: the communication with the pulmonary artery bore the impress of non-recency.

C.—COMMUNICATION WITH THE RIGHT VENTRICLE.

856. A man, aged twenty-five, in his ordinary state of health, felt a "crack in the heart," and became faint and pale, while lifting a sack of flour. Though very ill, he continued at work for three or four days. Nine weeks later, Dr. Hope found the face bloated and purplish, the legs very, the hands slightly, œdematous, the pulse 80, and excessively jerking; no pain; the least effort causing dyspnœa and irregularity of pulse for two or three minutes. The following physical signs also existed—thrill in the third interspace,[4] two inches from the sternum, with very loud superficial sawing murmur, like a whispered *r* at the same spot, most marked in systole, less in diastole, with also a continuous rumbling sound—no thrill nor murmur above the clavicles, impulse of the heart not materially increased. The patient died, highly anasarcous, nine weeks after the examination. A sac, as large as a small hen's egg, immediately above the aortic valves, opened by two small apertures into the right ventricle.

In a male, aged thirty-one, observed by Mr. Beck, both sounds are represented to have been distinctly audible; and immediately following, and loudest just after, the second sound, a sawing murmur disagreeably close to the ear, accompanied with thrill, most distinct at the base of the heart near to the sternum—but at which side of that bone is not stated—was heard.[5]

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[1] *Vide* an interesting one by Dr. Hughes Bennett.

[2] Dublin Journal, vol. xviii.

[3] U. C. Museum, No. 2254.

[4] Although this is not stated in the narrative, the left interspace is evidently meant;—Diseases of the Heart.

[5] Med.-Chir. Trans., vol. xxv.

D.—COMMUNICATION WITH THE RIGHT AURICLE.

857. There is reason to believe that where communication is established between an aneurism of the arch and the right auricle, the attendant thrill and murmur will be of maximum strength in the second and third right interspaces.

Little, if anything, is known of the duration of life subsequent to this perforation. An ostler, aged thirty, was brought in dead to University College Hospital at about half-past ten P. M.; that evening he had been heard to groan by a fellow-workman, and answered to inquiries as to the cause, that "he should soon be better;" after this, having taken some tea and a small quantity of gin, he fell in the street while returning to work at about ten P. M. and died almost immediately. An aneurism of the ascending aorta, three inches in diameter, had opened by a rupture, jagged on its aneurismal, smooth on its auricular surface, into the right auricle—when opened out, the communication seemed about as large as a fourpenny piece.[1]

This man had been known to have suffered for a long time from pain in the chest, increased by stooping; still he was looked upon by those who knew him as a healthy person: the aneurism alone, it seems evident, did not seriously disturb him. The rupture was, in all probability, partially effected at the time of the groan, completed at the moment of the fall.

E.—COMMUNICATION WITH THE LEFT CARDIAC CAVITIES.

858. I have not met with any example of communication by rupture between an aneurism of the aorta and the ventricle or auricle of its own side of the heart. Either occurrence, especially the former, is indeed difficult enough of conception.

§ VI.—COARCTATION AND OBLITERATION OF THE AORTA.

A.—ARCH OF THE AORTA.

859. The arch of the aorta is subject to a process of closure, more or less complete, either as a result of imperfection in the developmental changes occurring immediately after birth (quasi-congenital variety), or as an effect of disease in adult life (acquired variety).

I.—QUASI-CONGENITAL VARIETY.

860. The closure of the ductus arteriosus, commencing after a few respirations, is, according to Bernt, completed in some cases by the third or fourth day, by the eighth in half the number, and by the tenth in all. Now this closure normally exercises no influence on the contiguous portion of the arch of the aorta; but in a certain

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[1] U. C. Museum, No. 4026, described by Mr. St. John Edwards, "Medical Times," Dec. 1851.

number of instances the contracting process abnormally extends thereto.[1] The constriction has commonly been found at, or close to, the ductus arteriosus; varies in length, but, unless in very exceptional instances, averages scarcely more than a line or two, as though a string had been tied round the part; varies also in amount from more or less considerable narrowing to complete obliteration. In the latter case the vessel presents the appearance of a ligamentous cord; in the former, the internal surface is, except in the rarest instances, smooth. Vegetations, when present, must greatly intensify murmur. The vessels springing from the arch are always more or less, sometimes enormously, dilated; and various second and third-class arteries, especially the deep cervical, intercostal, and epigastric similarly widened. Local dilatations, in addition to the general enlargement, simulate so many aneurismal sacs—as has particularly been seen in the intercostal arteries.[2]

Congenital deficiency of the aortic valves, and irregular distribution of the ulnar and certain other arteries have been witnessed.

861. In respect of symptoms, past experience divides these cases into four classes: a class in which no special symptoms attracted attention during life, and the constriction has been a *post-mortem* discovery; a second, in which symptoms of disease of the heart existed, or were supposed to exist; a third, wherein disturbances of the circulation were more or less confidently referred to an imaginary aneurism of the arch; and a fourth in which the physical signs, conjoined with slight symptoms, led to the true diagnosis.

Separating what is wholly contingent and accidental from the real functional effects of constriction, the following symptoms may be admitted.

Fits of dyspnœa, dry irritable cough, and occasional hæmoptysis occur, but only when the heart itself is unusually excited, or under the influence of more or less violent strain on the circulation. Uneasy sensations may be experienced within the chest, probably both in the aorta itself [810] and in the cardiac plexus, but actual pain is, at the least, rare.

As effects of impeded circulation in the brain, fulness in the head, cephalalgia, vertigo, and epistaxis occasionally present themselves.

There seems no disposition to stagnation within the systemic capillaries (just as in aneurism of the arch also); even œdema of the feet fails to occur, unless as a sequence of intermediate obstruction in the lungs and right heart. Neither does the urine become albuminous.

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[1] Objections have, however, been taken to this idea of the mechanism of the constriction by Dr. Chevers—also by Rokitansky, who adopts Reynaud's theory of dependence on perverted development of the branchial arches where continuous with the aorta.

[2] Vide Clin. Lect. on Aortic Coarctation, Med. Times, Oct. 1857; also fig. by Alex. Meckel, copied into Craigie's Memoir, Ed. Med. & Surg. Journal, Oct. 1841; also the opposite diagram.

The influence exercised on general nutrition seems less than might be anticipated; in the case, which I described some years ago, the four extremities, especially the lower, were very fairly nourished, a fact the more remarkable in regard of the latter as they lived wholly, or almost wholly, an *anastomotic life.*

Fig. 1.

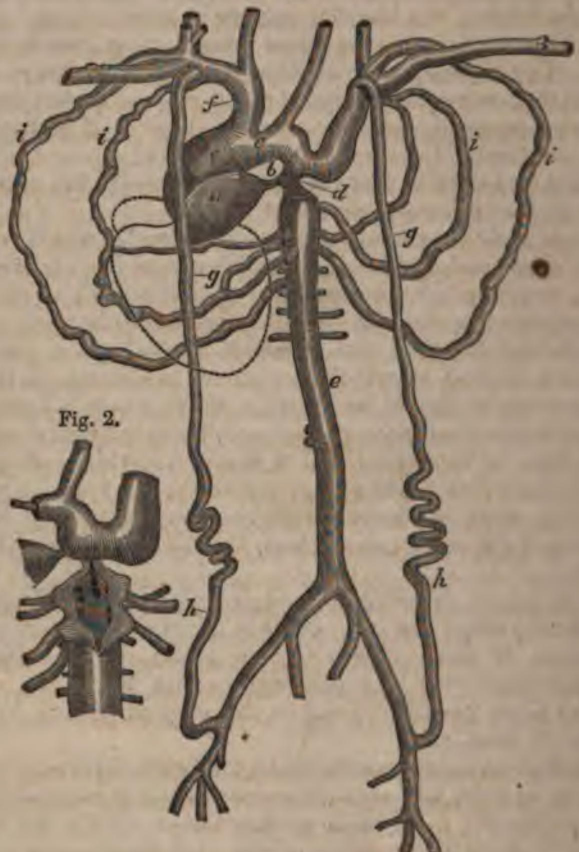

Fig. 2.

(*After an unpublished drawing by Carswell.*)

Fig. 1. *a*, pulmonary artery; *b*, arterial duct; *c*, arch of aorta; *d*, coarctation of aorta; *e*, descending aorta; *f*, innominate artery; *g*, internal mammary artery; *h*, epigastric artery; *i*, *i*, *i*, *i*, deep-seated arteries of neck and intercostals, forming, together with the internal mammary and epigastric arteries, a collateral circulation with the thoracic and abdominal aorta and internal iliacs.

Fig. 2. Aorta laid open, showing by probe the amount of constriction.

I do not imagine that a constricted state of the aorta is likely to generate or modify any diathetic state; tubercle in the lung has more than once been noticed, but, I presume, by accidental association only. Low inflammations, especially in the inferior extremities, might be theoretically looked for; but have not to my knowledge been found. The man, who fell under my notice, had slight lymphatic inflammation and whitlow in one of his upper limbs.

862. Not the least important and readily ascertainable of the physical signs, are presented by the extreme pulsation of the carotid, subclavian, temporal, deep cervical, and intercostal arteries. Occasionally local expansile impulse, aneurismal to the feel (and sometimes actually wearing away the ribs), may be felt from place to place in the latter vessels. Further, even at the top of the sternum, if the innominate artery and the aorta itself on the cardiac side of the constriction be much dilated, undue impulse may reach the surface.

Murmur may be heard of maximum intensity at the second right interspace close to the sternum; harsh; of sawing special character; high-pitched; conveyed upwards, to the right, to the left, downwards in the direction of both ventricles, but more to the right than the left, backwards to both vertebral grooves, more to the right than the left; persistent; not notably influenced by change of posture; in rhythm synchronous at its commencement with the close of the heart's first sound: such were the characters of the murmur in the case I have elsewhere described.[1] Probably generated by the passage of blood through the coarctation, it might possibly also depend on rippling of the current in the widened part of the arch. In this case the heart's own sounds were well audible.

There is no visible prominence at the top of the sternum.

Thrill may be felt at the second right, less at the second left, interspace, sometimes above the clavicles, and also in the site of the pulsating prominences of the intercostal vessels. Jugular pulsation of cardio-respiratory rhythm may be seen.

Either percussion at the top of the chest gives normal results, or, if the innominata and the arch on the cardiac side of the obstruction be much dilated, some slight unnatural dulness may be detected.

863. Of the causes indirectly leading to the coarctation nothing is known. It has hitherto been seen with greater frequency in males than females, in a ratio of about 3 : 1.

864. That quasi-congenital coarctation is not necessarily a malformation of fatal tendency, appears sufficiently from the fact that it has in eight or ten instances been found in the bodies of persons dying at upwards of fifty years of age, and in the case recorded by Reynaud the individual had actually attained the patriarchal term of ninety-two.

Yet on the other hand the prognosis is always uncertain; the annals of cases show that death may take place in the midst of robust health, with scarcely a moment's warning. The patient may perish in one of three main ways. First, he may be cut off suddenly (through the aortic affection) by rupture of the vessel itself, by rupture of the heart, and by formation and rupture of dissecting aortic aneurism.[1] One recorded case, at least (that of Meckel), shows that rupture may occur where habitual quietude of impulse seemed to render such a catastrophe a most unlikely event. Secondly, he may die of cerebral disturbance or pulmonary congestion, pneumonia or bronchitis, or of dilated heart—dependencies on the aortic obstruction. And, thirdly, he may be cut off by some completely unconnected, cardiac or other, disease.

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[1] Med. Times and Gaz., Oct. 1857.

865. If a systolic, or rather slightly post-systolic murmur, following the track of the aorta and of maximum force above the heart's base, were discovered as a permanent condition in a person free from spanæmia and from any of the ordinary murmur-producing affections of the valves, and if there were no local bulging, and at the most but slight abnormal dulness at the top of the sternum, while the patient suffered neither from the local symptoms nor the concentric pressure-signs of aneurism or mediastinal tumor, nor from cyanosis, there would be fair motive for strongly suspecting the existence of coarctation of the arch. This suspicion would be converted into certainty, if throbbing of dilated and sacculated cervical and intercostal arteries were more or less markedly present. But were this latter condition absent, the surety of the diagnosis would fail, inasmuch as all the signs previously rehearsed might depend on an obstruction at the aortic orifice of the innominate artery. For further details concerning the diagnosis, I would beg leave to refer to the clinical lecture already mentioned.

866. Curative treatment of a case of this stamp is of course out of the question. Nevertheless the part of the physician is by no means unimportant: symptoms, congestive and other, must be vigorously met, as they arise; and the occurrence of such symptoms warded off as far as possible by insisting on a careful system of hygienics. Now of all hygienic rules the most essential is that the patient avoid physical, emotional, and even intellectual excitement—that his life be a tranquil one. It is of extreme consequence, too, that he avoid exposure to cold and damp, and excesses of all kinds, such as must end in perturbed circulation and local congestions.

II.—ACQUIRED VARIETY.

867. The calibre of the arch of the aorta may undergo diminution both as a result of disease of its own, as also of certain affections of the heart, of the lungs, and of others of the system at large. Under the first head appear contractions depending on exudation-matter connected with the coats of the vessel. Under the second, range themselves reductions of size depending on the closely con-

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[1] Barker, Med.-Chir. Trans., vol. xliii. p. 131.

strictive action of induration-matter in the pericardium, and on obstruction of the mitral orifice, which entails a deficient supply of blood to the aorta. Next, prolonged obstruction of the pulmonary circulation acts in a somewhat similar way: the calibre of the aorta is below the average in persons cut off with vesicular emphysema of long duration, unless the right ventricle have become the seat of dilated hypertrophy. Lastly, in cancer and phthisis the vessel suffers in the same manner, probably from the gradual reduction in the amount of circulating fluid in those diseases.

B.—THORACIC AND ABDOMINAL AORTA.

868. A considerable number of cases are on record in which imperviousness, more or less absolute, of the aorta has been produced in the adult by coagula forming around prominent spiculæ of calcification, or by the products of inflammation, aided by contraction of the walls of the vessel itself. The lower part of the thoracic and the abdomical aorta are the most common seats of the disease.

869. The course of this affection, symptomatically, may be acute or chronic.

(*a*.) In some of the former class of cases, the current of blood, though it may long have been somewhat obstructed, has not become seriously interfered with until the appearance of acute symptoms—these symptoms being dyspnœa, anasarca, tendency to gangrene of the lower extremities, and hæmoptysis. Still, as the state of the lungs and heart is imperfectly known in the very few instances of the kind, the direct dependence of hæmoptysis on the obstruction may be questioned.

But occasionally the attack is to the last degree sudden in its graver manifestations. A merchant, aged twenty-nine, generally enjoying good health, is abruptly, after a few days' indisposition, seized with a burning sensation in the lower extremities, which rises towards the head; he lies almost deprived of consciousness. Shortly after he is found with a livid bloated countenance, and speechless; the left side almost deprived of motion, the head confused, and the pulse infrequent; when raised, vomiting at once supervenes. After the occurrence of a variety of well-defined local symptoms (stony coldness of surface, lividity, pulselessness of arterial trunks, tenderness of these on pressure, anæsthesia, motor paralysis, mummification of one lower extremity and moist gangrene of the other, the saphena vein being here also blocked up), all evidently dependent on obstruction to the arterial circulation, the patient perished on the 6th of December—the forty-sixth day from the acute seizure. The abdominal aorta, close to its bifurcation, was completely closed up by a firm, pale red coagulum, tightly adherent to the inner coat, which was smooth and pale. Plastic exudation existed between the membranes; acute inflammation of the arterial texture, as diagnosed during life by M. Romberg, was the evident cause of the plug-formation. Several other arteries were similarly affected. The difficulty of finding any even remotely probable cause is not the least curious part of this case.[1]

(*b.*) The chronic mode of progress is well illustrated by the following brief particulars of a case which I had the opportunity of observing in the wards of M. Louis in the year 1835. In this patient, a female, aged fifty-one, four years previous to death, numbness, first of the right, and, some months later, of the left, lower extremity, were clearly the earliest positive effects of the growing obstruction. These were followed by inability to walk, not from fatigue or cardiac suffering, but from pain, coldness, and increasing numbness in the legs. Subsequently, the effects of organic disease of the heart (she had mitral constriction, dilated hypertrophy of the right ventricle, palpitation, pulmonary apoplexy, and hæmoptysis) threw those of the aortic obstruction into the shade, and eventually cut her off in a few days after admission to the hospital. Twice in the course of her illness she had slight and passing œdema of the lower extremities, but there was none on her admission, and at death scarcely any. Besides a complete local obliteration above the iliacs, the calibre of the aorta generally, and of its branches, was below the average in this woman. No attempt appeared to have been made at anastomotic enlargements; the disease had, to all seeming, originated in inflammation of the vessel.

Dr. Gull records a very remarkable case, in which obstruction of the abdominal portion of the vessel having produced serious paraplegic symptoms, eventually, in proportion as anastomotic enlargements ensued, the motor paralysis materially improved.[2]

§ VII.—RUPTURES, PERFORATIONS, AND INJURIES OF THE AORTA.

870. Spontaneous rupture of the aorta has sometimes occurred, where, if observers may be implicitly trusted, the vessel was free from any form of pre-existing disease; more commonly more or less extensive calcification has been found; in rarer instances a softened state of the coats is referred to in descriptions.

Rupture appears particularly prone to occur in the ascending portion of the thoracic aorta. More frequently the fissure is found at right angles with the long axis of the vessel than in any other direction.

Of thirteen cases belonging to this category, eight were furnished by males, five by females.[3] Spontaneous rupture seems to be most frequent between the ages of thirty-five and fifty.

Death is not necessarily instantaneous; the external coat may not give way at first, and some hours, or even days may elapse, before a second rupture, breaking through this also, destroys life instanter.

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[1] Romberg, Dis. of Nervous System, vol. ii. p. 238.

[2] Guy's Hosp. Reports, 1857.

[3] Crisp, Dis. of Arteries, p. 289.

871. Ruptures from external violence are of rare occurrence, and almost limited to the male sex.

872. Ulcerative perforation of the vessel has in rare instances been observed, commonly as the result of a foreign body making its way from the œsophagus.[1]

873. Injuries to the chest-wall of various kinds have occasionally caused fatal rupture of the arch of the aorta. But a question of great interest is, to what extent physical shocks to the surface (especially about the second right cartilage, where the vessel most closely nears the parietes, and also especially in thin people) may inflict injury on the artery, without leaving any very notable trace on the integuments? I saw a case some years ago raising this question in an urgent form, in connection with a railway concussion;[2] and the inquiries made on that occasion revealed a total absence of fixed professional opinion, as well as extreme poverty of printed information bearing on the matter. Nor can I, to my regret, help in supplying the deficiency, except by venturing to direct attention to the subject. Here are the facts. A man, aged under forty (younger, consequently, than the average of sufferers from aneurism of the aorta), was to all seeming in perfect health, when he was thrown violently forward by a sudden jar of the train in which he was seated—the upper part of the right side of the chest pitching against an arm-rest between two seats opposite. It was deposed in evidence that this person had never complained of chest-symptoms, and that he had not consulted any medical man concerning the state of his heart or lungs. Now, within two hours after the accident the medical officer of the railway company concerned found a pulsating prominence in the neighborhood of the second right cartilage. This aneurism, for aneurism it was, eventually destroyed the sufferer in about eleven months after its discovery. The man pleaded that the aneurism was caused by the injury. The following arguments might be used in favor of his plea. 1. The chest-wall, where flexible, and in thin persons (among whom he ranked) is so easily depressed, that, as is well known, murmur may be produced by a very moderate amount of stethoscopic pressure. 2. The injuries to the thorax which have produced actual rupture of the aorta have ranged between violences the most terrible and so trivial, as to appear almost incapable of producing the effect. 3. Injuries to the chest-wall have occasionally ruptured the lung, without either fracture of a rib, or serious external marks of violence.[3] 4. Morgagni records several cases in which abdominal organs were torn by blows from sticks, though there was no injury to the skin, not even an ecchymosed stain.[4] 5. Numerous instances have been published in which a direct con-

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[1] Arch. Gén. de. Méd. 1825; Flower, Lancet, June, 1853.

[2] Case seen in consultation with Mr. Skey and Mr. Erichsen.

[3] Art. Emphysema, Cyc. of Surgery, p. 82.

[4] De Sed. et Caus. Morb. Ep. liv. § 15.

nection between external injury and aneurism of the arch has been traced and admitted.[1] 6. A case is recorded in which an aneurism was first perceived within twenty-four hours after the receipt of a blow with the fist on the right side of the chest.[2]

Now conceding that all these arguments put together do not constitute proof in the affirmative, they show that it would be difficult enough to maintain a negative. It seems to me well conceivable, that when calcification has pre-existed to any extent in the vessel, an injury of the kind described might lead to sudden breakage of the lining and middle, and blood-distension of the external, tunics.

SECTION II.—CORONARY VESSELS.

§ I.—RUPTURE OF A CORONARY ARTERY.

874. An individual, subject to palpitation, had a sudden attack of retching, with cardiac anguish, and died in an hour. The left coronary artery, widened, hardened, and fragile, was broken across from the aorta; effusion of blood had taken place between the pericardium and great vessels.[3]

§ II.—ANEURISM OF A CORONARY ARTERY.

875. This is a very rare affection in a genuine form; while fusiform dilatations of the origin of the vessel, and widening of its general tract, are far from rare.

The sac has, in the few instances on record, been included in the substance of the ventricle or of the auricle (almost always on the left side). It has contained coagula, sometimes distinctly laminated and partially decolorized.[4]

In some of the few cases death has actually resulted from, or been hastened by, rupture of the sac: where no rupture has occurred, no evidence exists that the affection gave rise to any symptom. It is noteworthy that in no single instance, though in some cases associated with calcification and general dilatation of the vessel, has aneurism of the coronary artery been said to have caused symptoms of angina pectoris—a fact which would almost require me to modify somewhat a statement, hazarded concerning the influence of the coronary arteries, in the history of the latter disease [428].

§ III.—VARIX OF THE CORONARY VEINS.

876. Sudden death has been occasionally caused by rupture of a varicose coronary vein. In two cases of the kind, observed by Albers, there had been symptoms of cardiac asthma, but evidently

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[1] *E. g.* Cases vii. x. xi. xii., in Dr. Crisp's collection, p. 150 et seq.

[2] Case vii. Crisp, loc. cit.

[3] Lombard, Gaz. Méd. de Paris, iii. 644.

[4] Peacock, On Aneurism of the Coronary Artery, p. 5.

traceable rather to the state of the heart itself than of the dilated vein.[1]

SECTION III.—PULMONARY ARTERY.

§ I.—BLOOD CONCRETIONS IN THE PULMONARY ARTERY.

877. The pulmonary artery, like all the channels in the body for venous blood, is prone to suffer obstruction on a small or large scale from the coagulation of its contained fluid.

878. The conditions leading to this coagulation are very similar to those traceable in the analogous case of clotting within the heart's cavities [772]. They may be arranged as follows: (*a*) blood-alteration, of which the essential tendency is to increase the coagulating property of the fluid; (*b*) some form of constitutional state rendering the circulation more sluggish than natural; (*c*) local causes of obstruction within the vessel concerned.

(*a*.) The blood-conditions are specially hyperinosis, the peculiar crasis in parturient women, and spanæmia.

(*b*.) All forms of debility, whether traceable directly or not to the influence of disease, past or present, fall under this head. In the convalescence of acute diseases, particularly those which present other favoring conditions, it is probable slight coagulation may happen pretty often, while it is certain that the process occurs on a serious scale in a small proportion of instances. Pneumonia supplies an illustration.[2] There is, I think, motive for believing that "relapse" is occasionally constituted, not by real return of the disease, but by blood-clotting within certain branches of the pulmonary artery; while it seems to me there can be no reasonable doubt protracted convalescence, and long continuance of dulness under percussion, with some form of bronchial breathing, are sometimes due to such coagulation. Syncopal tendency, as is familiarly known, is another condition which dynamically promotes the clotting-process.

(*c*.) The local aids vary with the position in the vessel where the separation of the fibrin takes place. In one class of cases the minute branches are its primary seat, whence it gradually extends to the larger trunks. Here, either pressure-obstruction, on the outside, by nodulous blood-infiltration, by exudation-matter, by adventitious deposits, and by infiltrating growths—or obstruction by a plugging process within the minute ramusculi, lays the groundwork. The plugging agent may, in the latter case, be derived from materials, fibrinous or exudative, borne along from the right side of the heart directly, or be furnished by minute fragments, separated from coagula formed in some distant part of the venous system, and carried along until they reach portions of the vessel too narrow to allow of further onward movement. This is a mechanism rendered, *à priori*, probable by the special minuteness of the pulmonary capillaries, as well as by the experiments of Cruveilhier and others, which long since showed that various foreign bodies introduced into the venous current stagnate in the minutest vessels of the lung. This mode of production has of late received general support: but obviously it can play no part where, on the one hand, there is no evidence of coagulation in any other near or distant part of the venous system; and where, on the other, the clotting process is limited to the larger branches of the pulmonary artery. In this form of case, where the larger branches are alone implicated, local aid to stagnation is found in the abruptness with which the artery divides into a number of branches, and in the numerous angular projections into the current at their points of bifurcation.[1]

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[1] Br. and For. Med. Rev., July, 1845.

[2] Louis, in Baron, loc. cit. p. 17.

Although disease of the inner coat of the pulmonary artery, inflammatory or other, may act, and has occasionally acted, as the local cause of obstructed current, yet instances of the kind are singularly rare; nothing can be more clearly demonstrated in the entire group of cases of venous stagnation than the habitual absence and consequent non-necessity of primary vascular inflammation.

879. *Anatomical appearances.*—These clots are either uniformly fibrinous looking on the surface, or of variously mottled colors and appearances—arising out of mixture of hæmatin and of changes of tint which this has undergone. They may be almost black on the surface; generally speaking firm, sometimes remarkably so, and smooth on the outside, they are usually short—not reaching in the large branches beyond the ramification immediately next to that in which they mainly formed. Clots may coexist in various parts of the vessel—several independent sources of coagulation being thus simultaneously, or nearly so, at work. The distal end of the clot from the heart is generally more or less fined off; but this appearance may be, and often is, wanting in small-sized coagula—which terminate abruptly and, as it were, shapelessly. The clot is generally adherent; sometimes throughout its whole length and periphery, more commonly by a limited spot only. The adhesion may be sufficiently firm to cause some difficulty in removal; so that the lining membrane undergoes some very slight superficial injury in the operation.

Provided the patient be not at once destroyed either by the clotting process mainly, or by this added to pre-existing disease, various changes may occur in the coagula, more or less identical with those observed in other parts of the venous system. Central softening may take place; and probably prelude disintegration and more or less effective solution and removal. Or the coagula, growing firmly to the wall of the vessel, may harden, and in this condition undergo more or less complete absorption, leaving behind them various residues, filamentous, lumpy, or stratiform. There seems reason to believe that some at least of so-called stony concretions in the lungs have been nothing more than phleboliths of the pulmonary artery.

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[1] Humphry on Venous Coagulation, p. 29.

In the small branches complete and permanent obliteration may occur.

Sequential mischief to the coats of the vessel is not observed; no vascularity of the lining, nor exudation into the external, membrane. At least this is very certainly the rule; but that in some rare instances of death, not really obstructive and of unintelligible mechanism, the fatal event may have depended on sequential pulmonary arteritis, seems by no means unlikely.

880. *Symptoms.*—The symptoms vary extremely with the site, the size, and the rapidity of formation of the coagula. In this clinical point of view, cases may be conveniently thrown into three groups.

881. (A.) When the clotting process is on a limited scale and occurs within branches of small calibre, no definite symptoms can, practically speaking, arise. At least the remnants of coagula, evidently of old formation, have occasionally been found, where the lungs had not been suspected to be the seat of any long-past mischief.

I am not aware that the existence of such old-standing obstructions in the branches of the pulmonary artery has ever been diagnosed; but I once saw a case, presenting the following peculiarities, which possibly might be explained on such hypothesis. A middle aged man, suffering from dyspnœa and chronic cough, free from the ordinary signs and symptoms of chronic consolidation, emphysema, tubercle, bronchitis, and the graver class of pulmonary and cardiac diseases, presented a strong blowing murmur, following the direction of the pulmonary artery, on the left of the sternum, inaudible at the apex and also at the base of the heart, extending well across and into the left axilla and to the costal angles, but not to the actual vertebral groove. On three examinations, at intervals of about a fortnight, this murmur presented precisely the same characters. I could not divest myself of the idea that it probably originated about the main branches of the left pulmonary artery—and if so (as there was no condition present to cause pressure on the vessel from without) partial internal obstruction seemed to afford the most likely explanation.

882. (B.) In a second class of cases the amount of coagulation is considerable; while the process, slow at first, eventually, and with variable abruptness, rapidly advances. As might be expected, this mode of progress is reproduced in the course of the symptoms.

The essential effect is dyspnœa, more or less marked, but commonly intense, the patient crying out for air; to this are added prostration, faintness, or actual syncope; tumultuous movement of the heart, or action scarcely differing from the usual type; pulse at the wrist feeble to actual deficiency almost; if venesection be performed, scarcely any blood flows from the vessel; sense of tightness at the præcordia; turgid lividity of the face; œdema of the extremities, with partially violet discoloration of their surface; great general distress, jactitation and dread of dissolution, while the encephalic functions remain unaffected.

Total absence of ordinary acute cardiac and pulmonary affections may be satisfactorily substantiated, provided the patient's condition permits of the necessary examination being made. In some cases (but, as *post-mortem* examinations prove, not in all) the physical signs of accumulation in the right cavities of the heart may be expected to occur [456, 774]. The only physical evidence connected with the vessel itself would probably be systolic basic murmur, following the course of the pulmonary main trunk, and of its immediate divisions to the left and the right of the sternum. This sign I most certainly heard in the case of an aged gentleman, whose life was brought to a sudden close, in the course of an acute affection, by coagulation in the pulmonary artery, and to a moderate extent, in the right ventricle. The murmur in question can, of course, be only expected to exist where the large trunks are involved; of physical evidence of deep-seated acute coagulation in the small branches I know nothing by experience. Perhaps some such murmur as that described a moment since from the history of a chronic case might be detected [881].

In almost all carefully recorded cases belonging to this category notable relief from the first anguish has been experienced; recurrence, more than once, of paroxysmal suffocation having taken place before the final struggle.

It appears, then, that the influence of coagulation in the pulmonary artery on life varies exceedingly. Not only may the formation of clots, even of good size, in the larger branches not destroy life at the time, but they may not sensibly shorten existence. Or such formation may bring life to a very sudden, though not actually instantaneous, close, out of its unaided influence; or it may, in a variety of morbid states, hasten an issue, which but for it might, perhaps, have been indefinitely averted. And under all circumstances, were the diagnosis established, the prognosis becomes to the last degree grave: we have no right to expect that in any particular instance the course of events shall place the patient in the exceptional category of those in whom the site and relationships of the coagula save them from fatal results.

883. *Treatment.*—The intense turgidness of the external venous system suggests the advantage of relieving the stasis by blood-letting; but when tried no such relief has been obtained—in truth, the blood has not flowed with freedom. The probabilities are, too, that anything like a copious flow, which certainly might lessen the evils of venous plethora for the moment, would eventually place the patient in a worse position than before. In cases running a lingering course, dry cupping of the chest, the application of Junod's boot to a limb, mustard poultices to various parts of the surface,

with free use of diffusible and alcoholic stimuli, afford the best chance of maintaining the circulation. If rally occur from the first urgent effects, the management of the case should be conducted in the manner recommended for coagulation within the heart [779].

884. (C.) A third class of cases is characteristically represented in the following brief narrative:—

> "A healthy female, aged thirty-eight, naturally confined of a fourth healthy child (lochia moderate, milk-secretion artificially stopped, without inconvenience of any kind), appears in the most satisfactory condition possible on the thirteenth day after delivery. After a good night's rest, she makes a light breakfast in bed on the fourteenth day, spontaneously remarking how thoroughly well she feels. She then rises (for the first time), chats with people around her, while her hair is being dressed, when suddenly, without warning of any kind, she cries out, 'Oh, my God!' her head falls on one side, and she instantly expires.
>
> "Sectio cadaveris the following day; weather cold. No appearance of putrefaction; cadaveric staining posteriorly. Encephalon strikingly pale, its arteries empty. Lungs remarkably healthy-looking. Heart small; no notable sub-pericardial fat; from right cavities a large quantity of blood escapes, coagulated, to various amounts, without homogeneous or laminated clot, that in, the auricle being of the consistence of partially melted jelly, that in the ventricle (reaching somewhat into the pulmonary artery) being somewhat firmer. Aorta empty, calibre below usual par, healthy. Heart's tissue somewhat flaccid right side, contracted firmly left; carefully examined, microscopically, is found perfectly free from fatty metamorphosis.
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> "The contents of the thorax being next removed, very fluid blood pours in abundance from the venæ cavæ, a state of fluidity which, with one exception was strikingly evident throughout the entire venous system. Right pulmonary artery empty; left contains coagula of gradually increasing consistence as the branches become smaller; thus, in the fourth or fifth divisions, the clots are thin, resisting, elastic, but colored shreds. The periphery of the clots, which are non-laminated in the larger branches, is firmer than their centre; nowhere can a central nucleus be found. Uterus firm, size of a large fist; the veins in its substance contain but little blood; those passing out of it, on the contrary, are distended by very slightly clotted blood, like that in the right auricle, as are also the inferior cava, the iliacs, and the femorals, as far as the external saphena. The most careful search fails to detect any particle of fibrinous coagulum."

These particulars, very kindly furnished me by Dr. Guéneau de Mussy, typify a class of cases, of which a considerable number have been observed among females of late years. The essential features of the group are these; recent parturition (generally not a fortnight past); natural and healthy progress after delivery; slight effort; posture sitting or standing; sudden cry, unpreceded by the slightest uneasiness of any kind, followed by instantaneous death, so instantaneous in some cases as to be assimilable to that produced by section of the medulla oblongata. The body is opened; and nothing found to attract attention except coagula in the pulmonary artery, associated or not with coagula in other portions of the venous system.

885. The interpretation of the facts, commonly adopted, ascribes the fatal event to abrupt stoppage of circulation in the lungs, by the clotting-process in their arteries. That this process actually commences in the large branches or trunk is held by some observers;

while others maintain the plugging theory, the necessary fibrinous plugs for the minute vessels being furnished by coagula in some distant vein.

The question of precedence in the sites of coagulation is of altogether subsidiary importance; yet it may be as well to observe, that careful examination has failed in several instances to detect any formed fibrinous coagulum within the veins of sufficient tenacity to justify the belief that fragments would have borne the journey through an extensive venous tract without reduction to a state of deliquescence and friability. Independent clotting within the pulmonary artery is in such cases the only legitimate hypothesis.

But wherein lies the proof that the clotting-process is really the cause of death in these terrible cases? In several instances of the kind the coagula have been found in medium-sized and small branches only, they have not filled the bore of the vessel, they have even (as in the instance just narrated) been limited to the artery of one lung. I can conceive fully, as already shown, that death may be effected and rapidly effected, by the formation of clots in the pulmonary main trunks. But even then instantaneous death is not to be looked for, the formation of such clots cannot be itself the work of an instant of time; and, if not the work of an instant, why should not the attendant clinical phenomena of death be asphyxial? For my own part it seems to me most rational to look upon these deaths as primarily and essentially syncopal, the clotting-process being in some cases a possible coincidence, in the majority a sequence, and in all promoted by the plastic crasis of the blood in ex-parturient women. The immediate manner of death points emphatically to syncope; and further the brain is pale, its arteries empty; is this the state to be expected, had death really arisen from pulmonary arterial obstruction?

The causes of the syncope, in turn, are beyond our ken. In the greater number of cases the fatal event has occurred in the erect or sitting posture, oftentimes immediately upon some emotional excitement; while further, in more than one instance, the victim has suffered for a greater or less length of time from mental disquietude and anxiety.

886. There is commonly no time either for diagnosis or for treatment. In point of fact, the patient has usually been either actually or well-nigh dead, when medical aid has been obtained. Should life not be extinct, the treatment to be adopted, on the instant, is that for syncope [405].

887. The story of the manner, in which existing knowledge on the subject of spontaneous coagulation generally has been acquired, is not a little curious. It adds one more to the numerous examples of the resuscitation, in the guise of novelties, of old ideas. The facts are referable to two orders—those bearing on the coagulation-

process, and those particularly referring to the wedging-process of detached fragments of clot.

Now among modern observers M. Legroux has an unquestionable claim to first mention. In 1827 he published two cases of instantaneous gangrene of the hand and forearm, presumedly caused by the instantaneous obliteration of the brachial artery by a fragment detached from a coagulum in the heart; he further generalized the idea of the migration of particles of clot through the entire arterial system.[1] Next appear the observations of M. Louis (1837), of M. Lediberder (1837), and of M. Baron, whose essay, incorporating cases by all three, was printed in 1838.[2] Then follows the evidence given by Dr. John Taylor in his Lectures on Pericarditis, showing that exudation-products, borne from the right ventricle into the pulmonary artery, might effectually plug its minute branches.[3] After this appears the contribution of Mr. Paget, the earliest general account in our language of the coagulation-process in the pulmonary artery.[4] Next in order stand the observations of Dr. Kirkes, on the lodgment in the cerebral vessels of fibrinous particles borne through the circulation from the left side of the heart.[5] Lastly, M. Virchow, in applying the old terms thrombosis and thrombus to the coagulation-process and its result, and in inventing that of emboli, to signify particles of detached coagulum, acting as vascular plugs, has afresh directed attention to, and popularized, the entire subject.

But the original statement of these ideas, especially of the more striking among them (that of vascular plugging), is to be found, as pointed out by M. Legroux himself,[6] in the Aphorisms of Boerhaave, and also in the Sepulchretum of Bonetus. A passage from the former author's lucid review of the causes and mechanism of apoplexy so completely and succinctly embodies the whole doctrine, that I cannot abstain from quoting it. "Quæcumque *sanguinem*, lympham, materiem spirituum ita mutant, *ut per arterias cerebri libere ire non possint, sed impacta hæreant:* talia sunt sæpe—*Polyposæ* in carotidibus, vertebralibusque arteriis *concretiones, sive circa cor primò*, sive intra ipsum cranium, *factæ*."[7]

§ II.—INFLAMMATION OF THE PULMONARY ARTERY.

888. We have seen that in cases of coagulation within the pulmonary artery, as a class, inflammation of the coats of the vessel is very rare. It is rare not only as a primary condition, but even in that sequential form which pretty frequently follows on the clotting-process, when occurring in the veins of the extremities.

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[1] Thèses de Paris, 13 Aout, 1827.

[2] Archives Gén. de Médecine, Mai, 1838.

[3] Lancet, 1845—orally earlier.

[4] Med.-Chir. Trans. 1844.

[5] Med.-Chir. Trans. 1852.

[6] Journal Hebdomadaire, 6 Nov. 1857.

[7] G. van Swieten, Comment. in H. Boerhaave Aphoris. (Apoplexia), t. iii. p. 258. Ed. Parisiis, 1771.

And further it appears very distinctly, that authors, who have insisted much on the comparative frequency of inflammation of the pulmonary artery, have assumed its existence wherever firm fibrinous coagula were found, especially if associated with sanguineous discoloration of the vessel.[1]

But such coagula and such discoloration by no means prove that inflammation has occurred:—*post-mortem* staining has evidently been confounded with true hyperæmic injection. In a word, examples of genuine inflammation, demonstrated by lymph-exudation within the tunics of the vessel, or on its inner surface, with fibrinous [illegible] around the external coat, are very difficult to discover. These conditions, combined with capillary sub-epithelial injection, roughness and loss of polish of the lining membrane, constitute the anatomical characters of the disease.

These appearances may be found in certain divisions of the main trunk,[2] or they may be limited to the valves, as in an instance recorded by the late Dr. Graves.[3]

889. Running an acute or sub-acute course, this inflammation is alleged to have been observed especially in pyæmia and certain other diseased conditions of the blood, in pneumonia, as a sequence of phlebitis, uterine or other, in Bright's disease, and as an idiopathic (?) state. But the actual evidence tendered of its existence is often anything but convincing, as may be easily ascertained by perusing the essay of Dr. Chevers.[4]

890. No attempt has been made, so far as I know, to establish a definite physical diagnosis of the disease—and never having seen a satisfactory case of the kind, I am unable to supply the deficiency. The probabilities obviously are that physical signs would arise rather out of the clotting process attending the inflammation than out of the changes in the vessel itself.

So, too, the symptoms in alleged inflammation have been identically the same as those arising as a consequence of rapid, but not instantaneous, coagulation, without the intervention of an inflammatory process. To the outline of these symptoms, already given, the reader may consequently be referred [882].

891. Treatment of inflammation of the pulmonary artery cannot be said to have ever been even attempted, seeing that the affection has never been diagnosed. And inasmuch as it forms an addition only to some profoundly grave pre-existent complaint, active measures can hardly be conceived warrantable. Were the diagnosis accomplished, counter-irritation and dry cupping of the chest, in

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[1] The cases of M. Cruveilhier (Anat. Path. livr. xi.), often quoted as examples of inflammation of the vessel, sequential to uterine phlebitis after delivery, possess no proven claims to rank in the category of inflammations. The central "pus" in the clots was obviously softened fibrin.

[2] L'Expérience, Mars, 1842.

[3] Dublin Journal, vol. xxii.

[4] Morbid Conditions of the Pulmonary Artery, p. 79 et seq.

association with the internal remedies advised in cases of coagulation, ought to be tried. The tendency to sinking calls for free exhibition of stimuli.

§ III.—ULCERATION OF THE PULMONARY ARTERY.

892. The rarity of ulcerative perforation of the pulmonary artery, on anything like a large scale, in the course of phthisis, is familiarly known. The branches of the vessel amid tuberculous deposit become clogged with coagula, so that even if a branch of some size undergo perforation, no hemorrhage ensues. The demonstration of the complete obliteration of the finer divisions of the vessel, amid and around tuberculous deposit, given by S. van der Kolk and Guillot, shows in a very satisfactory manner how it happens that destruction of wall may occur even extensively without giving rise to clinical disturbance.[1]

Still, both in the course of ordinary phthisis and of tuberculization of the bronchial glands, fatal hemorrhage may arise from perforation of a large branch, or, in the latter case, of the trunk, of the pulmonary artery.

893. In rare instances of abscess, and in more frequent ones of gangrene of the lung, a branch of some size, unprotected by coagula, is partially destroyed. The coagulating process seems to occur more sluggishly in the child than in the adult; and hence it is that hæmoptysis is relatively more common in childhood (a period at which its occurrence from all causes combined is, absolutely speaking, so rare) than after puberty, as an effect of sphacelated lung.

§ IV.—RUPTURE OF THE PULMONARY ARTERY AND ITS MAIN BRANCHES.

894. Rupture of the main trunk has been observed, as in the aorta, both from emotional excitement and under the influence of violent effort, whether the vessel were actually affected by or free from well-defined previous disease of its coats.[2]

Although death has been described as sudden in these cases, it does not appear to have been actually instantaneous in any of them. Mr. P. Hewett relates a case in which though the vessel was extensively ruptured, and the left auricle lacerated, the individual survived nearly four hours.[3]

895. And, where a main branch only is concerned, life may be much longer in ebbing away. A man, aged sixty-three, long the subject of bronchitis, the signs of which were alone discovered by auscultation, had a sudden attack of violent hæmoptysis. The bleeding, at first controlled, recurred, and death followed from exhaustion on the fourth day; the right pulmonary artery had under-

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[1] Louis on Phthisis, Syd. Soc. Trans., p. 29.

[2] Dr. Chever's essay contains examples of all those modes of occurrence.

[3] Lond. Med. Gaz., 1847.

cated by a permanent aperture.[1] Bronchitis, pneumonia, and hydrothorax seem, occasionally, sequential states.

899. The physical signs in indubitable cases have somewhat varied. The most satisfactory, actually observed, have been pulsating prominence, centralizing in the second left interspace, close to the sternum, and the seat of strong systolic thrill; and systolic, superficial, loud, harsh, rasping or grating murmur, of maximum force at the prominence, and followed, in one instance, where a pulmonary valve was contracted, by a very short diastolic murmur. But in Skoda's case there is not a single sign mentioned fairly referable to the aneurism; he himself supposes the absence of murmur explicable by the narrowness of the two main divisions of the vessel.

900. The prominent symptomatic states have been lividity of face, described as of cyanotic depth of tint in some cases, anasarca, ascites, great dyspnœa, cough and frequently recurring bronchitis, scanty urine, suppression of the catamenia, cephalalgia, and pain in the chest or at the epigastrium. The pulse, of variable frequency, is without special character.

But the most apparently significant symptom of the series may be wanting: Dr. Fletcher's patient was pale instead of being livid, though the two ventricles communicated besides.

901. Pulmonary aneurism has been observed in both sexes; in persons in the prime of life, and in girls under twenty.

902. Information is yet wanting as to the duration of life. In Skoda's case death followed the appearance of dropsy in three months; Dr. Fletcher's patient lived certainly twenty months—probably three years—with the disease.

903. The diagnosis of aneurism of this vessel may be considered tolerably certain, if pulsating prominence, limited to the left of the sternum and the second and third cartilages, furnishing strong, superficial, systolic, thrill and harsh murmur of the same rhythm—neither of them discoverable at the top of the sternum or above the clavicles—coexist with deep lividity of the face and anasarca, for which symptoms no ordinary explanation can be found. But aneurism of the ascending and transverse part of the arch of the aorta may protrude mainly to the left of the sternum [829 (*a*)]; and tumor in the mediastinum, pressing on, and receiving impulse from, the pulmonary artery, as also great hypertrophy of the left auricle, may simulate more or less closely these states; lastly, pulsating empyema must not be altogether forgotten.

II.—DISSECTING VARIETIES.

904. A soldier, in the pontoon service, aged 21 years, well able to discharge his laborious duties during the three months he had been in the army, in spite of slight dyspnœa, went to bed in good

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[1] Fletcher, Med.-Chir. Trans., vol. xxv.

health, Dec. 7th, having been employed in carrying wood during the day. In the night he was roused by violent pain to the right of the sternum, with great dyspnœa: all this subsided in a few minutes, leaving him pale, cold, anxious, and perspiring. In a short time nothing remained but præcordial uneasiness, slight palpitation, and feebleness of voice. Next day he seemed well, but at night complained of exhaustion: the face became pale, the surface cold, the pulse imperceptible, and in a very short time he expired. The pericardium contained a large quantity of coagulated blood: there were extensive calcareous concretions in the pulmonary artery, the inner coat of the vessel being separated from the elastic for the space of three inches from its union with the right ventricle, and torn into shreds, projecting into the cavity of the vessel, while further on the lining membrane was coated with fibrin. Just at the origin of the vessel appeared a hole the size of a fourpenny piece, through which the escape into the pericardium had occurred.[1]

§ VII.—ATHEROMA AND CALCIFICATION.

905. That atheroma of the pulmonary artery, more especially of its medium-sized divisions, is not infinitely rare, has been fully ascertained since the habit of slitting up the vessel in ordinary *post-mortem* examinations has grown common. But, except as affording a nidus for calcareous precipitation, this fatty deposit seldom does any demonstrable mischief. I cannot help suspecting strongly, however, that in some instances it aids in the production of pulmonary apoplexy; and to one indubitable case, where it was connected with grave and repeated hæmoptysis, otherwise inexplicable, I have referred in another place.[2] Calcification, certainly very rare, is generally associated (probably, in some measure, at least, as an effect) with hypertrophy of the right ventricle. Of its perilous character, when locally carried to an extreme amount, the case just related affords sufficient proof [904].

§ VIII.—CANCER.

906. In cases of cancer of the lung-substance, especially of the encephaloid species, it is not uncommon to find more or less extensive plugging of even large-sized branches of the pulmonary artery with coagulated fibrin and cancerous substance. The mechanism of the occurrence seems to be this: obstruction to the blood-current is first effected in minute branches by pressure from without of the cancerous substance infiltrating the adjoining parenchyma; next, the stagnating blood coagulates; thirdly, the development of cancerous blastema into cell-structure takes place within the meshes of the resulting coagula. As I have searched in vain for cancer-

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[1] Helmbrecht in Caspar's Wochenschrift, March, 1842.

[2] Diseases of the Lungs, 3d Am. ed. p. 329.

cells, and even for suspicious molecular matter in actual circulation with the general mass of the moving blood in cases of well-developed general cancerous contamination, and as other observers have not proved more successful, I cannot suppose that the appearance of firm encephaloid in the pulmonary artery can be due to the mere evolution of ready-formed solid elements entangled in the stagnating fluid. Nor, on the other hand, do I know of any evidence proving, or even tending to show, that perforation of the vessel by cancerous growth on its exterior, and subsequent vegetation in its interior, ever occurs, after the fashion frequently observed in the case of the bronchia.

The narrative apparently most strongly militating in favor of actual translation of cancer-cells through the general venous circulation, that I know of, has been reported by A. Wernher. A man, aged 22, first seen January 22, with a rapidly-growing encephaloid mass of the knee and tibia, is attacked on the 27th with sudden præcordial pain to the left, then to the right, of the sternum, without cough, palpitation, increased cardiac dulness, or murmur; pulse 140. On the following night several attacks of dyspnœa occurred, with scarcely perceptible pulse; on the third and fourth days, both, some pure coagulated blood was expectorated. Two or three days later the dyspnœa diminished, and the man appeared very much as before his acute seizure. Amputation was performed February 7th. Pyrexia, hæmoptysis, prostration, followed; on the 19th rigors, dulness at the posterior bases; and on the 24th, death from asphyxia and collapse. The iliac and crural veins, and their branches, as well as the cava, seemed normal, and free from coagula. The right pulmonary artery was almost filled with coherent cancerous masses; the left similarly obstructed, but to a less degree. In some spots the cancerous substance was incorporated with the vascular walls, though generally those walls proved perfectly healthy. The capillaries and pulmonary veins were quite free. The cancerous masses consisted almost throughout of large oval primary cells, with single or double nucleus, like those of the encephaloid in the leg. In the blood of the right heart, and of the ascending cava, similar cells were found; but none of the kind in any other blood. The lung-substance had been destroyed by gangrene in some parts, of which the pulmonary arterial branches had specially undergone obliteration.[1]

It must be conceded the discovery of formed cancer-cells in the blood of the right heart and cava inferior, and of these parts alone, carries with it serious significance as to the possibility, without, I think, positively demonstrating the reality, of actual cell-translation. Let me note, in passing, how distinctly the clinical history of the case strengthens the statement made in the account of pulmonary blood-concretions, as to the improvement which invariably takes place in cases of coagulative obstruction after the first urgent suffering [882]; and also how precisely it supports the assertion that the mode of death from such obstruction should be asphyxial and not syncopal [885].

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[1] Henle's Zeitschrift, Neue Folge, 5r. Bd., p. 109, 1854.

SECTION IV.—PULMONARY VEINS.

§ I.—INFLAMMATION.

907. Information is yet more deficient on the subject of inflammation of the pulmonary veins than of the pulmonary arteries.

908. Dr. R. Lee records a case, of which the following are the more essential particulars. A primiparous woman, aged twenty, is seized with rigors on the fifth day after delivery: these are followed by rapid pulse and dyspnœa with left thoracic and abdominal pain. Death occurred on the twenty-fourth day of the seizure. Both pleuræ and both lungs were inflamed. The trunk of the left pulmonary vein, near its entrance into the left auricle, was found plugged with a greenish, soft, firmly adherent clot of lymph extending into several of the principal branches. The lymph-clot appeared brightly scarlet on its outer surface when separated from the vein. The smaller branches, into which the solid lymph did not reach, were filled with pus [softened fibrin?], and in some parts coated with a delicate layer of yellow lymph.[1] A very similar case has been described by Mr. Adams.[2] Singularly enough, neither one nor the other of these observers adverts in any way to the condition of the pulmonary artery.

909. These narratives furnish no clue to the future diagnosis of the disease.

§ II.—DILATATION.

910. The most noteworthy example of this state, I know of, has been recorded by Dr. Townsend, of Dublin. An emaciated man, aged sixty-one, who had for upwards of a year mainly suffered from paroxysmal dyspnœa (attended with percussion-dulness of the chest, and notably relieved by bleeding), free from rigors, night-perspirations or diarrhœa; with a pulse of 100, feeble and very soft, but regular; the cardiac movements and sounds excesssively weak; the respiration null over the left lung, exaggerated superiorly, but very weak inferiorly, in the right organ; dies asphyxiated after five weeks' stay in hospital. The lungs, emphysematous anteriorly, were excessively heavy and fluctuated distinctly. On cutting into the left lung three pounds and a half of blood poured forth. The pulmonary veins furnishing this blood reached at least four times the normal calibre, from the smallest radicles to the large trunks—the latter forming capacious sacs outside the auricle. The same condition, less developed, affected the veins of the right lung. The dilatation had arisen from pressure of tuberculous infiltration of the

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[1] Med.-Chir. Trans., vol. xix. 1835.

[2] Dublin Journal, vol. xiii. p. 142, 1841.

wall of the left auricle on the veins at their line of union with that cavity; so extreme was the pressure, that a sound could with difficulty be introduced through the veins into the auricle. The right cavities of the heart (otherwise healthy) were dilated. The bronchial glands had undergone extensive, the lungs very slight, tuberculization.[1]

§ III.—RUPTURE.

911. A man, aged twenty-one, perishes in a fit of hæmoptysis, after three years' suffering from symptoms of diseased heart. The pericardium was found adherent; both sides of the heart greatly dilated; the aortic valves obstructively diseased to a grave amount. The immediate cause of death was traced to a rupture, nearly an inch long, of one of the left pulmonary veins, close to its point of issue from the lung; the left pleural sac was distended with blood.[2]

§ IV.—CANCER.

912. The trunks and medium-sized branches of the pulmonary veins occasionally contain, in cases of cancerous disease of the parenchyma, more or less commingled fibrinous coagula and carcinomatous substance. This accumulation may occur in association with, or independently of, a similar state of the pulmonary artery.

Such obstruction of the interior of the vessel, if to any notable extent, must greatly increase the dyspnœa arising from infiltration of the parenchyma; but I am unacquainted with any means by which obstruction of the kind could be diagnosed—and more especially by which it could be distinguished from that much more common condition, obliteration of the veins by pressure of cancerous substance on the outer surface.

§ V.—BLOOD-CONCRETIONS.

913. Dr. Edward Smith has placed on record the case of a lady, aged twenty, who, being in apparently excellent health, and within a few days of her expected confinement of a second child, while lying, dressed, on the bed, suddenly uttered a shriek, threw her arms about, crying wildly for air, then became calm for a moment, saying she was "better," and expired. At the moment of death the face was deeply livid and the body bent. Forty hours after death: slightly sanious fluid had escaped from the nostrils; posterior vibices; no swelling of the legs; blood black and universally fluid, "except in the *pulmonary veins*, where the whole tube was filled by a cylinder of coagulum, not strongly adherent to the lining membrane of the vein." Heart flaccid, left cavities empty, right ventricle containing, right auricle distended with, black fluid blood; valves healthy. Arteries preternaturally small; no blood in any of them. Veins universally distended, their calibre about as much above as that of the arteries below par. No venous or

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[1] Dublin Journal, 1832.

[2] Portal, Mém. de l'Acad. des Sciences, Paris, 1784.

arterial rupture anywhere. Stomach and intestines enormously distended with gas. Lungs collapsed, but faintly crepitant. Sinuses and large cerebral veins full; nothing abnormal in the brain. The white corpuscles are said to have been in considerable excess—but there is no evidence that the increase had exceeded that usually occurring in pregnancy. The absence of any mention of the pulmonary artery in the narrative is most singular—and, taken in connection with the loaded state of the right and empty state of the left cavities, increases very seriously the difficulty of interpreting the mechanism by which life was immediately arrested. The narrator is inclined to look upon the pulmonary venous coagula "as a cause and not merely an attendant occurrence of the death."[1] The case illustrates the truth that the phenomena of syncopal and asphyxial death are closely allied.

914. The pulmonary veins become the seat of murmur, continuous and remittent, in cases of intense spanæmia; this murmur is audible in the back on each side of the mid-dorsal spine,[2] and may simulate in front, about the basic region of the heart, intra-cardiac diastolic murmur [297]. But whether any such continuous hum could be traced in, and might lead to at least the surmise of, coagulation within the pulmonary veins, remains to be determined.

SECTION V.—DUCTUS ARTERIOSUS.

§ I.—PATENCY IN THE ADULT.

915. Drs. Barlow and Babington give the main particulars of a case in which cyanosis is referred to a patent state of the arterial duct in a woman cut off at the age of thirty-four. A variety of other intracardiac and arterial morbid states existed; and, in point of fact, the case appears to have been one rather of direct communication of the pulmonary artery and aorta in the site of the duct, than an example of openness of that canal. The effects would, however, be identical in the two cases. Not the least remarkable part of the matter is, that Dr. T. W. King appears to have diagnosticated a patent state of the duct some months before death. It is, unfortunately, not stated on what specific grounds the diagnosis was made.[3]

§ II.—ANEURISM.

916. A male infant died on the third day of its existence, with lividity of face, difficult respiration, stifled cry, small, weak, frequent pulse, but natural temperature of the surface. The ductus arteriosus dilated to the size and in the form of a cherry-stone, was almost filled with clots; a passage, sufficient to admit a crow-quill, remaining pervious. The rest of the body presented nothing remarkable.[4]

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[1] Case read before Med. Soc. of London.

[2] Davis, U. C. H., Females, vol. iv. p. 138, 1848.

[3] Path. Trans., vol. i. p. 55.

[4] Billard, Mal. des Enfans Nouveaux-nés, Paris, 1828.

APPENDIX.

Vide pp. 42, 43.

DIASTOLIC BASIC THRILL.

I HAVE within the last few days seen, with Dr. Reynolds, a case of very highly marked diastolic basic murmur, in which, although the patient cannot be said to be positively anæmic, a well-defined thrill attends the reflux through the aortic orifice. This diastolic thrill is better conducted upwards above the heart's base, than, as the connected murmur, downwards, towards the ensiform cartilage—a difference in the mode of conduction of tactile and audible vibration, analogous to that so frequently noticeable in the instance of vocal vibration and vocal resonance. It is not unworthy of note that though the murmur is extremely loud at the base, a perfectly well-defined second sound, without murmur, is heard at the left apex [149, 718, 855].

Vide p. 101.

DISTINCTION OF PERI- AND ENDOCARDIAL MURMURS.

The sign, referred to in the text, of increase of intensity of præcordial murmur on bending the body forwards, as distinctive of pericardial origin, is not to be implicitly trusted to. I omitted to mention in the proper place that I have known endocardial increased, and indubitable pericardial murmur decreased by this change of attitude. The normal pericardial rub, which is so frequently excited by excessive action of a healthy heart, sometimes actually disappears if the trunk be held forwards.

Vide pp. 136, 164.

HEART'S RHYTHMIC MOTION.

I would strongly recommend the reader, who may feel interest in the subject of cardiac rhythmical action, to study a very striking chapter in Mr. Herbert Spencer's "First Principles" (Chap. xi. p. 813) on the general philosophy of the rhythm of motion.

Vide p. 153.

HINDOO MANGO-GROWING.

The exact manner in which the jugglers proceed is this. Having had the ground to themselves all the previous night, they make their exhibition the following day. First they show a seed, plant it, put a great basket over the ground, remove the basket in so many minutes, and show the plant growing—cover again, remove again, and show the plant with the leaves—cover again, remove and show it with flowers—finally, once more repeating the process, exhibit it with fruit.

Vide pp. 158, 272, 326.

CONVULSIONS AND HEART-DISEASE.

Reference has been made in the places, above indicated, to the occurrence of muscular spasms, both clonic and tonic, in connection with acknowledged cardiac defect, either essentially dynamic, as in angina pectoris—or textural, as in fatty metamorphosis of the muscular fibre—or depending on malformation, as in cyanosis. In these cases the heart's disturbance is the main disease, the clonic and tonic spasms the accident.

Now, conversely, I believe that in some cases of genuine so-called idiopathic epilepsy, where the spasm and its attendants constitute practically the disease, the first appreciable link in the chain of events composing the paroxysm may be a perverted dynamic impression in the cardiac plexus. In a word, I think, I have seen cases in which the *aura epileptica* sprang from that particular mesh of nerves. In one instance in particular, long under observation, the patient connects a peculiar sharp pain in the heart-region with the outset of the fit—automatically carrying the hand to that region (as I have actually seen) at the moment of seizure, and describing the painful sensation on recovery. I must add that I have not during a given fit, thus initiated, found any disturbance in the heart's rhythm.

Vide p. 257.

SIMPLE SOFTENING OF THE HEART.

I have lately seen, with Dr. Harley, a gentleman, aged fifty-seven, of profoundly cachectic aspect, laboring under chronic pleuritic effusion, and excessively feeble cardiac action—the impulse being invisible, and the cardiac sounds faint to a degree, the accent at the apex inclining to the second of the two. These conditions led me to surmise the existence of fatty metamorphosis. The heart, however, as ascertained by Dr. Harley, proved free from that special charge, but had undergone true softening of primary and independent character. The naked-eye and microscopical appearances of the organ tallied closely with those in the case of an infant sixteen days old, recently published by E. Wagner. "The left ventricle was so soft, that on attempting to open it, it broke in pieces, and became a pulpy mass. The right ventricle was in a similar, or even softer, condition, but the auricle was normal. On putting some of the muscular tissue of left ventricle under the microscope, it fell to pieces so readily that it did not require to be teased out—fragments of muscular fibres were seen, resembling the smooth, spindle-shaped cells of involuntary muscle. They possessed no striæ, and were, for the most part, nucleated. Free cells and granules were also observed, but no fat or globules. The sarcolemma was, in some parts, thickened and œdematous. The substance of the right ventricle had a somewhat similar appearance to that found in the left. No striated fibres were anywhere to be seen."[1]

Vide p. 302.

AORTIC REFLUX AND SUDDEN DEATH.

At the time the above page was written, I was unaware that the tendency to sudden death in aortic reflux (a tendency I have constantly referred to orally at University College Hospital since 1855) had been made the subject of comment by any other person. I have since learned that the fatal disposition in question was known to Professor Chomel, and has been studied and described by M. Aran. Of the immediate mechanism of the instantaneous syncope, that destroys life, so little can be stated with even an approach to certainty, that it appears to me useless to undertake the discussion. No evidence exists showing that the event is traceable to sudden interruption of blood-supply to the coronary arteries.

Vide pp. 325, 326, 330, 331.

CYANOSIS.

The lightening in the tint of the skin after death in cyanosed children, is in a certain sense the counterpart of the change of color noticed in the dead bodies of people cut off in the algide stage of cholera.

Dr. Reynolds, in his recent elaborate and scientific work (*Epilepsy*, p. 230), contributes three examples of cyanosis attended with convulsions.

The argument of Fouquier, concerning the circulation of black blood in the fœtal skin, is of very doubtful value. Dr. Chevers (*loc. cit.*, p. 79), insists upon the want of facts proving that the blood of the fœtus in utero is less perfectly oxygenated than that in the arteries of the mother.

The excessive rarity with which an amount or quality of discoloration, fairly assimilable to that of true cyanosis, occurs as a consequence of organic obstructive disease of the right heart in the adult, has always proved a stumbling block in the way of those who support the theory of venous stasis, solely and exclusively, as the cause of congenital cyanosis. Dr. Chevers attempts, more ingeniously than convincingly it seems to me, to get over the difficulty, by suggesting that the special pliant extensibility of the capillaries, appertaining to new-born infancy, is required to insure the necessary dilatation. But on what ground does he set aside the well-known tendency of the capillaries and venous radicles to augment in calibre with advancing years? He refers to a case in which extreme narrowing of the pulmonary orifice in adult life was unattended with the slightest lividity (*loc. cit.*, p. 76).

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[1] Year Book for 1860, p. 31.

Vide p. 376.

EFFECT OF POSTURE ON ABDOMINAL MURMUR.

Not only is Dr. Stokes right in supposing that the disappearance in the erect posture of an abdominal aortic murmur, which is audible in dorsal decumbency, will not distinguish aneurism from tumor, but I am certain, from recent knowledge, that even in simple abdominal pulsation the same effect may be produced by that change of attitude.

INDEX.

The numbers refer to the pages.

A

ABDOMINAL diseases, various, diagnosis from aneurism, 378.

Abdominal murmur, posture affecting, 376, 412.

Accented second sound at pulmonary cartilage, 287, 402; why sometimes absent, 290.

Acephalocysts of heart, 283.

Adhesion, pericardial, physical signs of, 193; with hypertrophy, 195; with atrophy, 195.

Adhesions pleural, præcardial, their effects, 193.

Age, effect of, on position of heart, 23; on weight of heart, 25.

Albuminuria in cardiac disease, 244; in dilatation, 251; in tricuspid reflux, 292; not in hypertrophy, 227.

Amyloid degeneration, 276.

Anæmia, cardiac murmur of, 83.

Anæmia of heart, 168; cause of death? 168; of dilatation, 168.

Anastomotic life of limbs, 386, 390.

Anæsthesia, cardiac, 133.

Aneurism, percussion-dulness of, 109; resistance of, 110; species and varieties of, 345; and simple pulsation, how distinguishable, 337.

Aneurism of abdominal aorta, physical signs, 375; symptoms, 376; duration and mode of death, 377; prognosis, 378; diagnosis, 378; treatment, 380.

Aneurism of arch of aorta, sacculating, 346; local effects, 347; physical signs, 348; symptoms, 355; course, 363; modes of death, 363; repulsions and affinities, 364; diagnosis, 365; duration and prognosis, 371; treatment, 371.

Aneurism of heart, anatomy of, 307; causes of, 308; symptoms, 308; physical signs, 309; mode of death in, 309; treatment of, 309.

Aneurism of thoracic aorta, signs, 370; symptoms, 371; manner of death in, 371; diagnosis, 371.

Aneurism of valves, 309.

Angina pectoris, symptoms of, 156; terminations of fit of, 159; manner of death, 159; recurrence of, 159; duration of, 160; anatomy of, 160; predisposing causes of, 161; diagnosis of, 162; prognosis of, 162; nature and mechanism of, 163; treatment of, 164.

Antiphlogistic and stimulant methods, extreme, illogical, 192.

Aorta, arch of, topography of, 21; thoracic topography of, 24; hyperæsthesia of, 341.

Aortic pulsation, abdominal, symptoms, 336; physical signs, 336; state of vessel in, 337; causes, 338; diagnosis, 338; prognosis, 340; treatment, 340.

Aortic obstruction, murmur of, 90; not always conducted most loudly to right, 90; physical signs proper, 293; indirect signs, 293; pulse, 294; capillary systems in, 294.

Aortic reflux, murmur of, 92; physical signs proper, 294; indirect signs, 295; pulse, 295; effects on capillary systems, 296; sudden death in, 149, 302, 411.

Aortic valves, topography of, 20.

Aortitis, acute, physical signs, 341; symptoms, 342; diagnosis, 342; prognosis, 343; treatment, 343.

Aortitis, chronic, 343.

Apex-beat, altered by extrinsic disease, 31; by cardiac disease, 32.

Apex, elevation of, in pericarditis, how simulated, 186.

Apex, in pericardial effusion, 176.

Apnœa in fatty heart, 268; mechanism of, 268; in other conditions, 301.

Apoplectiform, nodules of heart, 210; seizures in fatty heart, 271.

Apoplexy, pulmonary, in mitral disease, 289; not in fatty heart, 269.

Arcus senilis in fatty heart, 274.

Arch of aorta, smallest dilatation demonstrable, 110.

Artificial murmurs, 102.

Arterial murmurs, organic, 112; hæmic, 113; coagulation, 114; dynamic, 114.

Arterial sounds in health, 110; double and single, 111.

Ascites in heart disease, 243.

Atheroma, cause of murmur? 112; of aorta, 344; of pulmonary artery, 404.

Atrophy, cardiac, concentric, 217; eccentric, 218; partial, 218; of lungs, senile, 402; valvular, 216.

Attitudes in aneurism of the arch, 355.

Attrition-murmurs, 95.

Aura epileptica and cardiac plexus, 410.

Auricle, left, topography of, 19; right, topography of, 19; left, hypertrophy of, 237; right, hypertrophy of, 237; right and aortic aneurism communicating, 384.

Auricles, dilatation of, 256.

Auricular impulse, 41; soniferous, 75.

Auscultation of heart, fitting posture for, 54; mediate preferable, 54.

Auscultatory percussion, 47.

Auto-audible murmurs, 124.

Axis of heart in dilated hypertrophy, 225.

B

BLOOD-ALTERATION as element of cardiac dropsy, 240.

Blood-accumulation, fluid, in heart, 170; affects percussion, 51.

Blood-collision in right ventricle cause of systolic murmur? 89.

Blood-concretions, intra-cardiac, post-mortem, 317; intra-mortem, 317; ante-mortem, 317; vegetation-form, 318; causes, 319; effects, 320; physical signs, 320; symptoms, 321; results of, 321; diagnosis, 322; prognosis, 322; treatment, 322; in pulmonary artery, 393; in pulmonary veins, 407.

Blood, changed composition of, cardiac murmur of, 83; nature of changes of composition, 84; coagulation of, murmurs of, 84.

Bloodletting in pericarditis, 188.

Blood-murmurs, 83; of diastolic time, how simulated, 295.

Brain, how affected by hypertrophy of heart, 229–233.

Brandy in pericarditis, 191.

Bright's disease, enlarged heart in, 28; how related to heart affections, 228; pericarditis of, 192.

C

CALCIFICATION, of aorta, 344; of heart, 259.

Cancer, cardiac, anatomy, 279; symptoms, 280; latent, 281; endocardial, 281; pericardial, 282.

Cancer and aneurism, how related, 365.

Cancer-cells, translated through blood-current, 405.

Capillaries of pericardium, dilated, causing friction sound, 175.

Cardiac paralysis, centric, 133; reflex, 133; intrinsic, through ganglia, 133; from exhausted contractility, 134.

Cardiac region, deep, 20; superficial, 20; form of, in health, 29; in disease, 30; integuments of, 29, 30.

Carditis, acute, forms of, 207; idiopathic, general, 208; partial, 209; secondary, 209.

Carditis, chronic, 209.

Cartilages, aortic, 21; pulmonary, 21; innominate, 21.

Cataract and heart disease, 231.

Cava, superior, communicating with aneurism of arch, 381.

Centre-point of heart, 20.

Cerebro-spinal symptoms in pericarditis, 181.

Cerebral state, in mitral reflux, 288; in tricuspid reflux, 291; in aortic reflux, 296; in cyanosis, 326.

Chloroform, affecting heart-ganglia, 134; how kills, 150; inhalation in pseudo-angina, 167.

Chorea, intra-cardiac murmur of, 85.

Churning murmur, 96.

Cirrhosis, of heart, 87, 282; of liver and tricuspid reflux, 291; of lung, how displaces heart, 315.

Clicking pericardial murmur, 96.

Coagulation within heart, endocarditic, symptoms of, 200; treatment of, 204.

Coagulation-murmurs, intra-cardiac, 85; in arteries, 114.

Coagulation spontaneous, *vide*, pulmonary artery.

Coarctation of arch of aorta, *quasi*-congenital, anatomy, 384; symptoms, 385; diathetic states in, 385; physical signs, 387; causes, 387; prognosis, 386; diagnosis, 388; treatment, 388; acquired, 388.

Coarctation of thoracic and abdominal aorta, acute, 389; chronic, 390.

Concentric hypertrophy, 235.

Conduction, horizontal, of percussion-sound, 48.

Conduction of aortic reflux murmur, 92.

Congestion of the heart, active, 169; passive, 169; mechanical, 169; encephalic, in dilatation, 252; spinal, 252.

Continuous venous murmur, 119.

Convulsions and heart disease, 158, 272, 326, 410.

Cord, spinal, in fatty heart, 272.

Cordæ tendineæ, rupture of, 313.

Coronary arteries, in angina, 160, 259.

Coronary arteries, rupture of, 392; aneurism of, 392.

Coronary veins, varix of, 392.

Cramp, cardiac, 135; in angina, 164.

Crumpling pericardial murmur, 96.

Cyanæmia, nutrition in, 324; and tubercle not antagonistic, 329.

Cyanosis, symptoms, 324; physical signs, 326; period of origin, 328; duration, 328; manner of death, 329; anatomy and mechanism, 329; causes, 332; diagnosis, 332; prognosis, 333; treatment, 333; why not in ordinary heart disease, 331, 411.

D

DEAFNESS in aortic aneurism, 362.

Death, mode of, influence of, on heart's position, 24.

Death, from chloroform, 150; sensations attending, 152; signs of, 153; from angina, 159, 161; from anæmia of heart, 168.

Death, in acute pericarditis, 183.
Death in acute endocarditis, 204.
Death, mode of, in fatty heart, 273.
Death, mode of, in aneurism of heart, 3[illegible]9.
Death, in pulmonary coagulation, mechanism of, 405.
Decumbency, flat, in hydropericarditis, 179.
Decumbency in cardiac dilatation, 249.
Depression of cardiac region, 29.
Detrusion of heart, 314.
Diastolic apex murmurs, left, 91; right, 92; ditto, basic murmurs, aortic, 92; pulmonary, 94; never artificial, 103.
Diastolic arterial murmur, vessel healthy, 337.
Diastolic impulse, 40; double impulse, 40.
Diastolic valvular thrill, 42, 43, 409.
Diatheses, in diagnosis of pericarditis, 186; various and fatty heart, 264.
Diathetic diseases, atrophy of heart in, 27.
Digitalis, cardiac paræsthesia from, 133; how slackens heart's beat, 138; hazardous in dilatation, 254; in valvular disease, 306.
Dilatation of heart, from anæmia of its substance, 168; from congestion of ditto, 170; anatomy, 244; causes, 245; general, physical signs of, 247; symptoms of, 248–252; diagnosis of, 252; prognosis of, 253; treatment, 253.
Diphtheria, infrequent pulse after, 139.
Displacement of heart, affecting its sounds, 65.
Dissecting aneurism, of aorta, anatomy, 380; symptoms, 380; treatment, 381; of pulmonary artery, 403.
Divergence of stream, cause of murmur, 80, 85.
Double impulse, diastolic cardiac, 40; aneurismal, 350.
Dress, influence of, on position of heart, 60.
Dropsy, cardiac, situations of, 238; mechanism of, 239; treatment of, 242.
Dropsy, rarity of, in aneurism, 334.
Ductus arteriosus, in constricted arch, 384; patency of, in adult, 408; aneurism of, 408.
Dulness, præcordial, superficial and deep, 46; deep-seated cardiac, how affected by disease, 53; præcordial, movable, 53; of aneurismal sacs, 351; smallest demonstrable, 110.
Duration of endocardial murmurs, 81.
Dynamic murmurs, intra-cardiac, 85; arterial, 114.
Dynamic heart disease, characters of, 127–129.
Dysphagia in pericarditis, 181, 187; in aneurism, 356.
Dyspnœa, not essential to angina, 158; cardiac, paroxysmal, treatment of, 255.

E

Effusion, pericardial, quantity of, annulling friction-sound, 99; chronic pericardial, 196; diagnosis of, from dilatation, 253.
Elevation of heart, 315.
Emaciation, aneurismal, characters of, 355.
Emphysema, with visible pulses, 106; influence of, on mitral murmur, 89.
Endocardial murmurs, special character of, 80; rhythm of, 77; pitch of, 77, 81; distance of, from surface, 77; force of, 77, 80; duration of, 77; rhythm of, 77; how affecting heart's sounds, 78; influenced by posture, 79; species of, 78; organic causes of, 79; inorganic, 83; hæmic, 83; dynamic, 85; temporary, 86, 87; intermittent, 88; of individual orifices, 87–95; relative frequency of, 94; associations of, 95; distinction of, from pericardial, 101–104.
Endocardial murmur caused by pericardial lymph, 104; by pleural effusion, 104.
Endocardial murmurs, new and old, how distinguishable, 201.
Endocarditic murmur, usual permanency of, 204.
Endocarditis, acute, anatomy of, 197; physical signs, 198; local symptoms, 200; general symptoms, in free cases, 200; in obstructive cases, 200; causes, 201; diagnosis, 201; prognosis, 204; treatment, 204.
Endocarditis, chronic, anatomy of, 205.
Endo-pericarditis, acute, frequency of, 206; prognosis of, 206; chronic, 207.
Epigastrium sinking with heart's systole, 32.
Epileptiform seizures in fatty heart, 271.
Exudation-matter, pericarditic, affecting percussion, 53.
Exudation-matter, pericardial, without friction sound, 184.
Expansile pulsation demonstrated, 349; simulated, 340; of extravasated blood, 376.
Eyeballs in chronic palpitation, 143.

F

Fakirs, self-hypnotism of, 153.
Fatty infiltration, sub-pericardial, symptoms of, 260; sub-endocardial, 261.
Fatty metamorphosis, relation to dropsy, 239.
Feeble action of heart, 156.
Fibrin-plugs of minute vessels, 393, 397.
Finger ends in cyanosis, 325.
First sound, characters of, in health, 55; mechanism of, 58; duration of lessened, 68.
Flux, cardiac, intestinal, 243; gastric, 244; renal, 244.
Fœtal endocarditis, effects of, 198.
Foramen ovale, patent, cause of murmur? 327.
Foreign bodies in heart, 334.
Fremitus, vocal, in cardiac region, 42.
Friction-fremitus, pericardial, 43; distinctions from pleural, 43.
Friction sound, simulated over heart, 31; in pericardium, 95–104; in pericardial liquid effusion, 177.
Fusiform aneurism of arch, 368.

G

Galvanic current, influence of, on heart in syncope, 149, 155.
Ganglia of heart, 134.
Ganglionic system, effect of, on heart's motion, 133.
Gas in pericardium, 214, 215.
Gastric symptoms in pericarditis, 180, 186.
Globular aneurism of arch, 369.
Gout and fatty heart, 264.

H

Hæmic arterial murmurs, 113.
Hæmic intra-cardiac murmurs, species of, 83.
Hæmo-endocardium, 211.
Hæmo-pericardium, varieties of, 210; treatment of, 211.
Hæmoptysis in mitral reflux, 289; in coarctation of arch, 385.
Hemorrhage, aortic aneurismal, 359, 364; in cardiac dilatation, muscular, 249; gastric, 250; pulmonary, 250, 255; uterine, 251; intra-cardiac, 210.
Healing rupture of heart, 311.
Heart, situation of, 17, 20, 24; regions occupied by, 18; axis of, *in situ*, 17; base and apex of, how related to surface of chest, 17, 18; centre point of, 20; how related topographically to lungs, 20; actions of, clinically, 22; situation of, affected by age, 23; by dress, 23; by position, 23; by mode of death, 24; locomotion of, respiratory, 24; bulk of, normal, 25; thickness of walls, 26; cavities of, normal capacity of, 26.
Heart's nutrition, influence of, on valvular murmurs, 297.
Heart's sounds. *Vide* Sounds.
Horizontal conduction of percussion-sound, 48.
Hum, venous, 119.
Hydropericarditis, locular, simulating tumour, 185.
Hydropericardium, active, 212; passive, 212; mechanical, 213; symptoms of, 213; treatment of, 213.
Hydruria, in angina, 159; in cardiac disease, 244; in dilatation, 251.
Hyperæsthesia, cardiac, neuralgic, 130; tactile, 131; special, 131.
Hypertrophy, percussion in, 50; muscular, of heart, varieties and anatomy of, 219; micrology of, 221; causes of, 222; left ventricular, signs of, 223; symptoms of, 225; secondary lesions, 228; prognosis, 232; treatment, 233; concentric, 235; right ventricular, 236; signs of, 236; auricular, 237.

I

Impulse, of heart, visible, 30; in disease, 31; maximum transferred to base, 32; pendulum-like, 33; palpable, 34; synchronous occurrences, 34; clinical characters of, 35; modifying influences, 36; mechanism of, 36; in non-cardiac disease, 37; in cardiac disease, 38; auricular, 41; pseudo-auricular, 41; undulating, 33; horizontal, increase of, 39; diastolic, 40.
Impulses, cardiac, successive, normally equal, 40.
Induration of heart, 259.
Induration-matter, pericardial, effect on heart, 218.
Infrequent action of heart, mechanism of, 138.
Infrequent pulse, 138; in fatty heart, 269.
Inhibiting nervous influence on heart's motion, 138, 139, 150.
Innervation of valves perverted, cause of murmur, 86, 87.
Innominate artery, topography of, 21; aneurism of, 368; veins, continuous murmur in, 119; systolic single murmur in, 124.
Intensity of heart's sounds increased, 67; lessened, 67.
Intercostal arteries dilated in constriction of aorta, 387.
Intermittent mitral murmur, reflux, 88; obstructive, 91.
Intermittence of pulse, relation to syncope, 137.
Interspaces, præcordial, form of, 41.
Iodine injection into pericardium, 196.

J

Jugular vein, distension of, 115; more on one side than on other, though of cardiac origin, 115; murmur in, 119.

K

Kidney, movable and aortic pulsation, 340.

L

Laryngeal symptoms in aneurism of arch, 357; in thoracic aneurism, 370.
Laryngismus in pericarditis, 181.
Latent, abdominal aneurism, 376; endocarditis, 201; pericarditis, 182; wound of heart, 335.
Leucohæmia, venous murmur in, 122.
Liver, enlargement of, affecting heart's apex, 31.
Lungs and heart, how related topographically, 20.
Lymph, pericardial, raising apex-beat, 32.

M

Malformations of the heart, without symptoms, 323.
Malpositions of heart, congenital, 313; acquired, 314.
Mango-growing trick, 154, 410.
Mediastinal tumor simulating hydro-pericarditis, 185; diagnosis from aneurism, 367.

Memory, in fatty heart, 271.
Mensuration, as applied to præcordial surface, 38.
Mercury in pericarditis, 190.
Metamorphosis, fatty, infrequent pulse of, 139; relation to dropsy, 239; anatomy of, 262; micrology of, 262; chemistry of, 263; coexistences of, 263; nature of, 264; predisposing causes, 265; latency of, 266; physical signs of, 266; course of, 272; mode of death in, 273; diagnosis, 273; treatment, 275.
Mitral constriction, murmur of, 91; physical signs proper, 289; indirect signs, 290; pulse and capillary systems, 290.
Mitral murmur, auto-audible, 124.
Mitral regurgitant murmur, character of, 87–90.
Mitral regurgitation, physical signs proper, 287; indirect physical signs, 288; pulse, 288; effects on capillary systems, 288.
Mitral valve, topography of, 21, 25.
Mitral valve, competence of, how proved post mortem, 287.
Mixed aneurism of arch, 369.
Murmur, endocardial, in hypertrophy, 224.
Murmur, chronic endocardial, quoad prognosis, 302.
Murmurless aneurismal sacs, 353.
Murmurs, aneurismal, characters of, 353; audible at distance, 354; auto-audible, 124; coexistent at same orifice, 95; intracardiac caused by dilatation, 248; producible by palpitation, 143; wanting in mitral constriction, 289.
Murmur-like sounds, 70, 71.
Murmurs changing in site, acute, 199, 203; in rhythm, chronic, 299; endocardial, conditions modifying, 298; of acute endocarditis, 199; wanting in, 203.
Muscular contraction, soniferous, 60.
Myo-carditis. *See* Carditis.

N

NEURALGIA of cardiac nerves, 130.
Neuralgia, cardiac, 163; of arms, 167; phrenic, 179.
Neurilemma, oil within, 263.
Normal tricuspid reflux, 292.

O

OBSTRUCTION and reflux alternating at an orifice, 285.
Œdema, of the heart, 212; acute, general, and aortitis, 342; pulmonary, in heart-disease, 243; not in constriction of arch, 385.
Orifices of heart, width of, 26; diseases of, *vide* Valves.
Orthopnœa in pericarditis, 179.
Over-frequent action of heart, 140.

P

PAIN of aortic aneurism, 355, 358.
Palpitation, effect of on endocardial murmurs, 82; caused by special hyperæsthesia of heart, 131; simple, symptoms of, 142; physical signs of, 143; duration of, 144; causes of, 144; treatment of, 145; irregular, symptoms of, 146; physical signs of, 146; causes, 147; prognosis, 147; treatment, 147; in cyanosis, 326.
Papillary muscles, disordered action of, causing murmur, 86.
Paracentesis pericardii, 196; in hæmopericardium, 211; in hydropericardium, 214; in wounds of heart, 335.
Paræsthesiæ, cardiac, 132.
Paræsthesia in valvular disease, 300.
Paralysis, cardiac motor, 133; in angina, 164.
Paraplegia in aortic aneurism, 362.
Partial reduplication of second sound, 74.
Parturition, recent and pulmonary clotting, 396.
Pendulum-like impulse, 33.
Percussion, cardiac, difficulties of, 45; sound in health, 46; superficial and deep, 46; how modifiable physiologically, 48; ditto, by horizontal conduction, 48; in non-cardiac disease, 49; in cardiac disease, 49; of aneurisms, 110; dulness of hydropericarditis, how simulated, 184; in dilated hypertrophy, 224.
Pericardial adhesions indirectly influence percussion sound, 51.
Pericardial agglutination, effect of on heart, 187.
Pericardial effusion cause of endocardial murmur, 80.
Pericardial fluid, how affects percussion, 51; its dulness, how distinguished from that of enlargements of heart, 52.
Pericardial lymph causing endocardial murmur, 104.
Pericardial murmurs, 95; varieties of, 95; site of, 96; intensity of, 97; maximum position of, 97; rhythm of, 97; pitch of, 97; superficialness of, 98; simulation of, 98; conditions modifying, 98; time of occurrence of, 98; duration of, 99; causes of cessation of, 99; redux, 100; anatomical conditions of, 100; how affected by previous pericarditis, 101; distinction of from endocardial, 101–104, 409; effects of stethoscopic pressure on, 102; caused by granulated liver, 104; auto-audible, 124.
Pericardial sac, topography of, 22.
Pericardial surfaces, separation of, soniferous, 63.
Pericarditis, acute, anatomical characters, 171; secondary lesions, 173; causes, 173; physical signs, 175; symptoms, local, 178; general, 179; course and mode of progress, 182; terminations, 182; duration, 183; diagnosis, 183; prognosis, 187; treatment, 187.
Pericarditis, chronic, varieties of, 193.
Pericarditis, tuberculous, 277.
Phleboliths of pulmonary artery, 395.
Phthisis, pericarditis in, 175; heart in, 218; and fatty heart, 264; foramen ovale in, 329.
Phwo...tt sound, nature of, 93, 217.

Pitch, of arterial murmurs, 112, 113; of artificial murmurs, 103; of blood murmurs, 83; of coagulation murmurs, 84; of dynamic murmurs, 85; of endocardial murmurs, 77, 81; affected by conduction, 81; of first sound, raised, 65; of mitral regurgital murmurs, 88; tricuspid ditto, 90; of aortic constriction, 90; mitral obstruction, 92; of murmurs, affected by conduction, 82; of pericardial murmurs, 97.
Pleural effusion simulated by enlarged heart, 221.
Pleural friction of cardiac rhythm, 101, 184.
Pleural friction-fremitus, distinction from pericardial, 44.
Pneumo-hydropericarditis, varieties of, 215; signs of, 215.
Pneumo-hydropericardium, 214.
Pneumo-pericardium, 214.
Pneumo-pericardium, percussion in, 50.
Pneumothorax, mechanical cause of hydropericardium, 213.
Posture, influence of, on position of heart, 23; affecting murmurs, 79; influence of, on jugular distension, 115; on venous murmur, 119; effect of, on aortic abdominal murmur, 376.
Præ-mortem murmur, 203.
Procidentia of heart, 317.
Pressure, effects of, on anatomy of pericarditis, 172.
Pressure, internal, effect of on murmurs, 104.
Pressure, stethoscopic, effect of, on murmurs, 102–104; effect of on pitch of pericardial murmur, 97; causing arterial murmur, 111, 113; double, 114; influence of on venous murmur, 119.
"Progressive muscular atrophy" of heart? 219.
Pseudo-apoplexy in fatty heart, 271.
Pseudo-angina pectoris, symptoms of, 166; diagnosis of, 167; treatment of, 167.
Ptyalism, pericarditis occurring during, 190.
Pulmonary artery, topography of, 21; blood-concretions in, conditions favoring, 393; anatomy of, 394; inflammation, primary or sequential, rare, 395; symptoms of, on small scale, 395; on considerable scale, 395; influence of on life, 396; treatment, 396; in cases of sudden death, 397; inflammation of, anatomy, 400; course, 400; diagnosis and treatment, 400; ulceration of, 401; rupture of, 401; general dilatation, 402; aneurism of, sacculated, anatomy, 402; physical signs, 403; symptoms, 403; duration of life, 403; diagnosis, 403; aneurism of, dissecting, case of, 403; atheroma and calcification of, 404; cancer of, 404.
Pulmonary artery and aortic aneurism communicating, 382.
Pulmonary artery and branches, probable murmur in, 395.
Pulmonary capillaries in mitral reflux, 288; in tricuspid reflux, 291: in aortic obstruction, 294; in aortic reflux, 296.
Pulmonary obstruction, physical signs pro per, 296; indirect effects, 296.
Pulmonary reflux, murmur of, 94; physical signs of, 297.
Pulmonary valves, topography of, 20.
Pulmonary veins, murmur in, 123, 408; inflammation, 406; dilatation, 406; rupture, 407; cancer, 407; blood-concretions, 407.
Pulsation, arterial, visible, healthy, 106: abnormal, 106; value in aortic reflux, 106; in constriction of the arch, 107.
Pulsation, venous, visible, 115–117; jugular, how affected by respiration, 117; ditto, palpable? 118.
Pulse, varieties of, 108; in angina pectoris, 157; in left hypertrophy, 226; in fatty heart, 269; of tricuspid reflux, 291; of aortic constriction, 294; of aortic reflux, 295; of pulmonary obstruction, 297; of mitral reflux, 288; of mitral constriction, 290; in aortic aneurism, 361.
Pulse and heart-sounds, relation in disease, 71—74.
Pulseless systoles of heart, 73.
Pulse-respiration ratio, in pericarditis, 180; in fatty heart, 273.
Pupils in aortic aneurism, 363; in abdominal ditto, 377.
Pus in heart's structure, 207–209.
Pyramidal percussion-dulness of pericardial fluid, 52.
Pyrexia, frequent pulse of, 140.

Q

QUALITY of percussion-sound over heart altered, 53; of heart's sounds modified, 67; of endocardial murmurs, 76, 81.
Quasi-congenital coarctation of arch, 384.
Quickness of heart's contraction, modified, 140.

R

REDUPLICATION of heart's sounds, 73; varieties of, 74; causes of, 74; modifications of, 74; partial, 74; simulated, 75: diagnostic signification of, 75; how affects theory of heart's sounds, 76.
Redux pericardial friction, 100.
Reflex perversion of heart's rhythm, 137.
Reflux, converted into obstruction, at an orifice, 299.
Reflux, aortic, second sound at left apex in, 64, 382, 409.
Regurgitation, aortic, visible pulses in, 107. *Vide* Aortic.
Reinforced heart's sounds beyond site of production, 65.
Renal region, percussion of, influenced by horizontal conduction, 48 *note*.
Resonance, vocal in cardiac region, 105.
Respiration, locomotion of heart through, 24; in cardiac region, 105; in fatty heart, 268; in aortic aneurism, 358.
Reticulation of aortic valves, cause of partial reflux, 93, 94.

Reticulation of valves, 216; nature of, 217; symptoms and signs, 217.
Rhonchus, by heart's action, 104.
Rhythm, cardiac, perverted, 136; dynamic, 136; of parts of heart, 138; in palpitation, 146; in angina, 237.
Rhythmic motion of heart, 136, 164, 409.
Rhythmic nerve-centres of heart, 136.
Rippling stream, cause of murmur, 80; of arterial murmur, 112; venous, 122.
Rupture of aorta, spontaneous, 390; from violence, 391; without notable injury to surface, 391.
Rupture of heart, anatomy of, 310; causes, 310; symptoms, 311; treatment, 312; partial ditto, 313.
Rustling sound over heart, 98.

S

SACCHARINE precipitation in heart? 276.
Saturnism, infrequent pulse of, 139.
Scurvy, hæmopericardium in, 211.
Second sound, characters of in health, 56; mechanism of, 62; imitated in disease, 64; extensively audible, cause of, 68; state of in mitral disease, 88; in aortic reflux, 93–94; in pericardial adhesion, 194.
Sex, influence of, on thickness of walls of heart, 26.
Sexual inclination, in dilatation, 251; in fatty metamorphosis, 270.
Silences of heart, normal length of, 55; in disease, 71.
Sinking in of chest with systole of heart, 32.
Sinuses, cerebral, murmurs in, 119.
Softening of heart, modes of origin of, 257; signs of, 257; symptoms, 258; diagnosis, 258; treatment, 258; micrology of, 410.
Somatic death, signs of, 153.
Sounds of heart, characters of, 54; length of, 55; properties of, vary in different spots, 55, 57; extent to which audible, 57; mechanism of, 57–63, 76; modification of, 64; affected by displacement, 65; morbid states of, 66; distance of from ear, 69; quality and pitch changed, 69; relationship to pulse, 71; changed rhythm of, 72; reduplications of, 73; how affected by murmurs, 78.
Spanæmia, cardiac murmur of, 84.
Spanæmia, causing thrill, 109.
Spanæmia, relationship of to venous hum, 121.
Spasm, cardiac, 135; in angina, 163.
Sphygmometer, 109.
Sphygmoscope, 109.
Sterno-clavicular ligaments, section of, in aneurism, 374.
Stomach, distension of, fatal through syncope, 149.
Sudamina, reaction of fluid of, 180.
Sudden death, in angina pectoris, 161; from impaction of clot in an orifice, 320; of cancer in ditto, 281; pulmonary coagulation with, 397; in aortic reflux, 302, 411.
Symbols of heart-sounds, 57.
Syncope, causes and mechanism of, 148; anatomical characters of, 150; symptoms, premonitory, 151; actual, 151; duration of, 152; diagnosis of, 152; prognosis of, 154; treatment of, 154; relation to intermittent beat, 137; in fatty heart, 271; fatal in angina, 161; in pulmonary coagulation, 398.
Syphilitic deposit in heart, 277.
Systemic capillaries in mitral reflux, 288; in tricuspid reflux, 291; in aortic obstruction, 294.
Systemic capillaries in aortic reflux, 296.
Systole, impulse synchronous with, 35.
Systole, arterial, 107.
Systolic, murmur at left apex, 88; at right apex, 89; basic aortic murmur, 90; ditto, pulmonary ditto, 91.

T

TEMPERATURE, local, affecting heart's beat, 136.
Temperature of face in aortic aneurism, 363.
Temporary intra-cardiac murmur, 86, 87.
Temporary murmur with fatty heart, 267
Terminal pericarditis, 175.
Tetanus, cardiac spasm in, 135; cardiac cramp in? 135.
Thrill, arterial, 109, 112; venous, 118; basic diastolic, 409; aneurismal, 350.
Thyroid arteries in cardiac hypertrophy, 221.
Thyroid gland, in chronic palpitation, 143.
Thyroid gland, in hypertrophy of heart, 227.
Traction of heart, 315.
Tracheotomy in aneurism of arch, 374.
Tremor, cardiac, 141.
Tricuspid obstruction, murmur of, 92; physical signs proper, 292.
Tricuspid reflux, murmur of, 89; jugular pulsation in, 116; effect of on brain, 232; on dropsy, 239; physical signs proper, 290; indirect signs, 291; pulse, 291; effects on capillary systems, 291; a normal state? 292.
Tricuspid valves, topography of, 21.
Tubercle, cardiac, 277; endocardial, 277; pericardial, 277.
Tubercle and aneurism, how related, 365.
Tuberculization and cardiac dilatation, 246.
Twisting sidewards of aneurismal sac, 370.
Typhoid (Peyerian) fever, heart in, 257.

U

UNDULATORY impulse, 33; pseudo, 33.
Uræmia, cause of cardiac murmur, 84.
Uræmic coma, diagnosis from syncope, 152;
Urine, in angina, 158; in hypertrophy, 227; in aortic aneurism, 361; in cardiac dilatation, 251; in fatty metamorphosis, 270.

V

VAGI nerves, influence of on heart's beat, 138, 139; gastric branches of, in angina, 158.

Valves, topography of, 20; in chronic endocarditis, 205; atrophy of, 216; hypertrophy of, 219; diseases of, functional effects of, 284; diseases of, physical diagnosis in general, 297; symptoms, 300; mortality, duration, and prognosis, 301; treatment, 304; malformation of, 323.
Valvular disease, tactile hyperæsthesia in, 131.
Valvular murmurs, various, relative frequency of, acute, 199; chronic, new and old, how distinguishable, 201.
Valvular murmurs caused by pericarditis, 176, 177.
Valvular thrill, characters of, 42; diseases causing, 43; questionable if diastolic, 43, 409.
Varicose aneurisms of arch, 381.
Varix, murmur of, 124.
Vegetations, endocardial, 318.
Veins, distension of, 115; murmur in, 118–124; various quality of, 118; characters of, 118; conditions modifying, 119; occurring in what vessels, 119; rhythm of, 119; accompaniments of, 120; mechanism of 120; blood-states with, 121; diagnosis of, 123.
Vena Cava, superior, topography of, 22.
Venesection lessening præcordial dulness, 49.
Venous current, soniferous in health, 120, 326.
Ventricle, left, topography of, 19.
Ventricle, right, topography of, 19.
Ventricle, right, hypertrophy of, cause of, jugular pulse, 116.
Ventricle, right, dilatation of, 256.
Ventricle, right, and aortic aneurism communicating, 383.
Vocal fremitus in cardiac region, 42.

W

Walking exercise, in angina, 159: in dilatation, 254; in fatty disease, 276; in aortic aneurism, 373; in dynamic palpitation, 148.
Weight of organs, in hypertrophy of heart, 228; in cyanosis, 326.
White patches of pericardium, 197.
Wounds of the heart, symptoms of, 334; physical signs, 335; treatment, 335.

THE END.

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